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Archives of Neurology and Psychiatry

Vol. 10 JULY, 1923 No. 1

THE DECEREBRATE RIGIDITY OF SHERRINGTON IN MAN

ITS RECOGNITION AND DIFFERENTIATION FROM OTHER FORMS OF TONIC MUSCULAR CONTRACTION

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LONDON, ENG.

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I. INTRODUCTION

For the analysis of the symptoms of nervous disease in terms of normal function, the fundamental importance of the experimental physiology of the nervous system can scarcely be overestimated. Unfortunately, the demands of clinical neurology rarely allow us the opportunity of studying at first hand the experimental side of neurology. Moreover, although the fruits of experimental research are available to us in the literature, yet the correlation of animal experiments with clinical observation is a matter of no little difficulty, and unless attempted with considerable caution is a ready source of error and confusion for the clinical observer.

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Nowhere in neurology are we more dependent on the experimental analysis of function than in the case of the motor activities of the nervous system, and nowhere has the physiologist more brilliantly served us. We cannot hope to understand the numerous and complex disorders of muscle tone and of movement without some insight into the normal nervous processes underlying the coordination of movement. The last decade has seen a steadily increasing desire on the part of neurologists to avail themselves of the inspiration to be drawn from the physiologic laboratory. However, this method of approach is as difficult as it is full of promise, and the careful study of some recently published descriptions of what purports to be "decerebrate rigidity in man" indicates that grave misconception exists in the minds of many clinicians as to the postulates to be satisfied before a diagnosis in these terms can be established. Indeed, the meaning of this term has been so modified and debased in some clinical writings that we see it applied to conditions having no relation whatever to this very clear-cut, experimentally produced state of the musculature.

It is therefore proposed in the present paper to discuss briefly the anatomic and physiologic factors concerned in the production and maintenance of decerebrate rigidity in animals, and thereupon to proceed to the clinical application of this knowledge. Finally, to illustrate the conclusions reached, a small series of cases will be discussed from the point of view of their possible relationship to experimental decerebrate rigidity.

II. ANATOMIC CONSIDERATIONS

The pioneer observations and the minute physiologic analysis of the rigidity we owe to the genius of Sherrington,[1] and during the years 1912 to 1920 a series of over twenty papers have come from the laboratory of Professor Magnus of Utrecht, recording beautiful researches which have considerably amplified our knowledge of the postural activities underlying the coordination of movement.

1. *Planes of Transection.*—The decerebrate animal is a mammal (rabbit, cat, dog, or monkey) in which the brain-stem has been divided in the region of the tentorium, between the anterior and posterior colliculi. In these circumstances the extensor groups of muscles enter almost at once into a state of heightened tone, which has provided the medium for Sherrington's classic researches on this phenomenon, and which he has named "decerebrate rigidity." He found that transections as far caudad as the pons had the same effect, but that as soon as the plane of section reached the neighborhood of the calamus scriptorius, the rigidity was abolished

1. Sherrington, C. S.: Cataleptoid Reflexes in the Monkey, Proc. Roy. Soc. **60**:411, 1897; Decerebrate Rigidity and Reflex Coordination of Movements, J. Physiol. **22**:319, Pt. 4, 1898.

and the musculature became flaccid. Thiele[2] found that successive coronal transections, passing from the anterior end of the brain backwards, resulted in the appearance of rigidity only when the plane of transection reached that of the posterior end of the thalamus. Magnus[3] confirmed this observation finding that rigidity did not develop until the section reached a plane bounded dorsally by the posterior limits of the anterior colliculi and ventrally by the region immediately caudad to the corpora mammilaria. On further coronal sections the rigidity persisted undiminished until the plane of entry of the eighth nerves

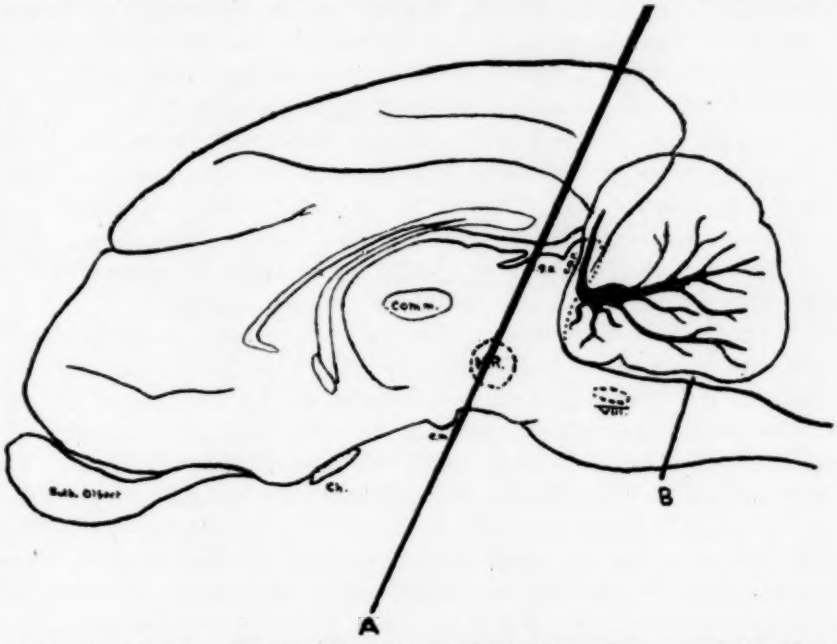

Fig. 1.—Median sagittal section of cat's brain (from Potter and Winkler's Atlas). *A*, transections cephalad to this transverse plane result in the production of an animal with tone of normal intensity. The animal can walk, jump and stand, and actively take up any posture on appropriate stimulation. *B*, transections of the brain-stem between the planes *A* and *B* result in the production of decerebrate rigidity or "reflex standing." The animal cannot right itself if overturned, walk or run. If any reflex change of posture occurs the animal falls. Transections caudad to the plane *B* abolish the rigidity and the musculature becomes flaccid.

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2. Thiele, F. H.: On the Efferent Relationship of the Optic Thalamus and Deiter's Nucleus to the Spinal Cord, with Especial Reference to the Cerebellar Influx of Dr. Hughlings Jackson and the Genesis of the Decerebrate Rigidity of Ord and Sherrington, J. Physiol. **32**:358, Pt. 5 and 6, 1905.

3. Magnus, R.: Welche Teile des Zentralnervensystems müssen für das Zustandekommen der tonischen Hals- und Labyrinthreflexe auf die Körpermuskulatur vorhanden sein? Arch. f. d. ges. Physiol. **159**:224, 1914; Beiträge zum Problem der Körperstellung. I. Stellreflexe beim Zwischenhirn- und Mittelhirnkaninchen, ibid. **163**:405, 1916.

was passed and the calamus scriptorius reached. Caudad to this, transection abolished the rigidity. These observations are best summarized in a diagram (Fig. 1) of a sagittal section of the cat's brain, taken from the Atlas of Winkler and Potter, in which these limits have been indicated.

2. *The Cerebellum.*—Contrary to the observations of Weed,[4] which in this respect we must now reject, Sherrington,[1] Thiele,[2] Horsley[5] and Magnus[3] have all found that extirpation of the cerebellum does not abolish decerebrate rigidity. In a large series of observations on the rabbit, cat, dog and ape, Magnus has finally established beyond any doubt that the cerebellum is not essential to the maintenance of decerebrate rigidity, which persists undiminished after removal of this structure and bilateral division of the eighth nerves.

3. *The Red Nucleus and Superior Cerebellar Peduncles.*—From these observations and the more recent ones of Bazett and Penfield[6] it follows that ablation of the red nuclei and division of both superior cerebellar peduncles do not abolish the rigidity. It is further clear that in the more caudad transections, the greater part of the pons and of the middle peduncles are also removed.

It is established, therefore, that the reflex centers concerned in the maintenance of decerebrate rigidity lie entirely in the pons and medulla, and that the afferent and efferent limbs of the arc do not pass through the cerebellum or its peduncles. In the cord these paths appear to lie in the ventrolateral columns, and such anatomic identification of the efferent path as is possible indicates Deiter's nucleus and the vestibulospinal tract as concerned in the reflex. As Sherrington has pointed out, the arc arises and ends in the muscles, for the tone of a given muscle depends essentially on the integrity of its afferent nerve supply.

III. PHYSIOLOGIC CONSIDERATIONS

The decerebrate animal is one from which the whole of the forebrain and 'tween-brain and the anterior part, or the whole, of the midbrain has been removed. In these circumstances all the muscles which maintain the animal in a standing posture are in strong tonic spasm. These include the limb extensors, the elevators of the head, neck and tail and the extensors of the spine, making up what Sherrington has called the "antigravity" muscles. The rigidity is an exaggeration of normal tone, and under the law of reciprocal innervation we accordingly find that the antagonistic flexor groups of muscles

4. Weed, L. H.: Observations on Decerebrate Rigidity, J. Physiol. **48**:205, Pt. 2 and 3, 1914.

5. Horsley, Victor: The Functions of the Cerebellum, Brain **29**:446, Pt. 4, 1906.

6. Bazett, H. C., and Penfield, W. G.: A Study of the Sherrington Decerebrate Animal in the Chronic as Well as in the Acute Condition, Brain **45**:185, Pt. 2, 1922.

are correspondingly inhibited and atonic. The head of the animal is upheld, the spine fully extended, the tail maintained horizontally and the limbs fully extended. The upper limbs are retracted at the shoulder and the forefeet held with the palmar surfaces directed ventrad as in standing. The hind limbs are similarly held. Such an animal, if carefully placed on its feet, can stand. It breathes automatically with a normal rhythm and shows numerous pseudaffective and other reflex reactions. Having no power of temperature regulation it cannot be kept alive for more than a few hours except with great difficulty in an incubator. Bazett and Penfield succeeded in keeping such animals alive for periods up to three weeks. Under these conditions, the rigidity is maintained throughout, though it is apt to be replaced from time to time by a plastic flexor rigidity. This receives some explanation in the light of Magnus and de Kleijn's observations to be referred to later. The study of the "chronic preparations" confirms the conclusions reached by Sherrington from his acute preparations as to the characters of decerebrate rigidity.

But although it can stand when passively erected, the animal can neither walk, run, nor jump, and if overturned has no power to right itself, but lies passively in the position determined by gravity. It may indeed take a single leap, but, unable to maintain its balance, it falls at once.

1. *Standing Reflexes.*—To appreciate fully the significance of this condition we must consider as a whole the nervous mechanisms by which posture is regulated.

In the course of his experiments, Sherrington [7] had observed that while the tone of a given muscle depended essentially on the integrity of its afferent nerve-supply, yet it was not wholly independent of afferent impulses from other regions. Thus, active or passive rotation of the head of the decerebrate animal on the neck inhibited the extensor tone in the limbs of that side of the body to which the vertex was turned. Still utilizing the decerebrate animal, Magnus and de Kleijn [8] found that by varying the position of the animal's head in relation to space and in relation to the neck and trunk it was possible to produce certain constant modifications in the tonus of the neck, trunk, and limb muscles, and thus in the posture of the animal. The existence of two groups of reflexes was determined, labyrinthine and neck reflexes. In each case the reactions are tonic in the sense that they are produced,

7. Sherrington, C. S.: Flexion Reflex of the Limb, Crossed Extension Reflex, and Reflex Stepping and Standing, J. Physiol. **40**:27, Pt. 1 and 2, 1910.

8. Magnus, R., and De Kleijn, A.: Die Abhängigkeit des Tonus der Extremitäten-muskeln von der Kopfstellung, Arch. f. d. ges. Physiol. **145**:455, 1912.

not by movement, but by the changed posture of the head, and they persist so long as the new posture is maintained, even though this be for months.

The labyrinthine reflexes arise from stimulation of the otolith organs of the labyrinths when the position of these is changed in relation to the horizontal plane. They reinforce the extensor tonus of the body musculature. The labyrinths affect all four limbs identically (identical innervation). After their destruction it is still possible to influence tone by varying the position of the head in relation to the neck and trunk (tonic neck reflexes). The neck reflexes arise in the proprioceptive nerve endings of the cervical muscles and joints. Thus, it is found that rotation of the head on its long axis, lateral flexion, dorsal flexion and ventral flexion produce constant changes in muscle tone and posture. Thus rotation of the head to the right (vertex to right, snout to left) causes increased extensor tonus in the limbs of the left side (the side to which the snout points, "Keiferbeine"). Lateral flexion of the head to the right causes increased extensor tonus in the right limbs and diminished or abolished extensor tonus in the left limbs. Hence, we see that alterations in the symmetrical relationship of the head to the trunk affect the limbs of the two sides reciprocally, and not identically.

2. *Position Reflexes (Stellreflexe) in the Thalamus and Midbrain Animals.*—The posture of the head (the leading segment, Sherrington) determines the attitude of the whole body (Körperstellung), and in the decerebrate animal we can vary the body posture at will, by using the head as a handle. However, we have only to consider the capacity of the normal animal to vary the position of its head and of its trunk at need, to realize that the reflexes we have so far studied are not the sum total of the postural reflexes active in the intact animal. The decerebrate animal possesses no reflex by which the position of its head can be actively modified. The head is unalterably retracted. Hence the animal can do nothing but stand. It is clear, therefore, that we must look in the higher levels of the nervous system for these postural reactions which the normal animal possesses and the so-called decerebrate animal lacks.

For this purpose Magnus has made use of the "thalamus" and "midbrain" preparations.[3] In an animal—rabbit, cat, dog, or monkey—the whole of the cerebral hemispheres, including the corpora striata are removed. The thalamus, subthalamic region, and midbrain remain intact. The midbrain preparation is one in which the thalamus also is removed, leaving part of the subthalamic structures and the whole midbrain intact. These preparations differ in but a single respect, namely, in the retention by the "thalamus" animal of normal tempera-

ture regulation, which the midbrain animal has lost. Although both these preparations are deprived of their cerebral hemispheres, yet they differ in a remarkable manner from the decerebrate animal with which we have so far been dealing. When the operation is completed, the animal (rabbit) is laid on its side, and as it recovers from the anesthetic it gradually rises to a squatting position, head first, and the neck and trunk slowly right themselves until the animal sits erect in a natural posture. The distribution of tone in the musculature is what Magnus describes as "normal." There is no extensor rigidity, and both extensors and flexors exhibit, as demonstrated by passive manipulation of the limbs, tone of normal intensity. Left to itself the thalamus or midbrain animal remains motionless, but it can be stimulated by auditory or cutaneous stimuli to movement—walking, running, or jumping. All these are carried out in a normal manner with perfect coordination, and when they cease the animal resumes its sitting position. If overturned it rights itself at once, and in whatever position the animal be placed, the head at once resumes the upright normal posture. The sum total of all these reflexes, Magnus designates position reflexes (Stellreflexe), as opposed to the tonic reactions described in the preceding section which he groups together as standing reflexes (Stehreflexe). For further details of these reactions the reader is referred to a recent review by the present writer in Medical Science.[9]

In the decerebrate animal all four groups of Stellreflexe are absent. Hence it is clear that the centers concerned lie in the midbrain cephalad to the plane of the posterior corpora quadrigemina. They persist after ablation of the cerebellum, and their afferent and efferent paths cannot, therefore, pass through this organ.

Observations on normal animals show that the tonic labyrinthine and neck reflexes and the position reflexes are the sum total of all the postural reactions of the intact animal.

We are now in a position to formulate the relative significance of decerebrate rigidity. The nervous mechanism concerned in the postural side of muscular coordination may be said to consist of two main elements, (*a*) of a reflex arc which by its activity maintains muscle tone. Tone is the basis, the raw material, of posture. Its adjustment in harmony with the various motor activities of the living organism is achieved by (*b*) a series of brain-stem centers and paths extending from the anterior end of the midbrain to the medulla. In the midbrain animal the whole of this regulating mechanism is in action. In the

9. Walshe, F. M. R.: The Work of Magnus and His Collaborators on the Nervous Regulation of Posture, and Its Bearings on Some Modern Neurological Problems, Med. Science **7**:109 (Nov.) 1922.

so-called decerebrate animal, however, the greater and dominant part of it has been destroyed. Such elements of it as are left can be thrown into action only by passive manipulation of the animal's head. It seems reasonable to suppose, therefore, that decerebrate rigidity represents the exclusive and unfettered control of the reflex arc whose activity maintains tone by the standing reflexes. This leads to an intensified tonic contraction in the muscles which keep the animal erect, and to a correspondingly great inhibition of tone in the antagonistic flexor groups. In short, the reflex mechanism which maintains tone is released not alone from cerebral cortical control, but also from that of the chief subcortical centers in the midbrain.

3. *Unilateral Decerebrate Rigidity.*—Since the clinical type of decerebrate rigidity most commonly found in man is, as we shall see, the spasticity of hemiplegia, it is clear that a consideration of unilateral experimental rigidity is essential. In his original studies on the subject, Sherrington found that hemidecerebration gave rise to homolateral extensor rigidity, which was a less constant phenomenon than the rigidity following complete decerebration. He also observed that stimulation of the intact excitable motor cortex could inhibit the rigidity. For example, in the case of the knee extensors, stimulation of a cortical point which gave rise to knee flexion inhibited the rigidity of the knee extensors. Therefore, the cortical influence on decerebrate rigidity is a crossed one, a fact not readily reconciled with the occurrence of homolateral rigidity on removal of one hemisphere. Later, Thiele found that in the more anterior hemidecerebrations, extensor rigidity was crossed, and he concluded that its appearance was due to removal of an influence descending from the thalamus and travelling in a path which decussated in the region of the rubrospinal decussation. Weed also confirmed Sherrington's observation that extensor rigidity was homolateral after hemidecerebration, and like this observer was able to inhibit it by stimulation of the intact crossed cortex.

Bazett and Penfield's chronic hemidecerebrate preparations have provided the solution of this paradox, for it is found that after an initial phase of homolateral extensor rigidity, a crossed extensor rigidity develops and persists as long as the preparation survives. The initial phase they regard as due to operative stimulation, the second and permanent phase as representing true reflex decerebrate rigidity due to "release." There are other points of interest in their observations with which we are not at present concerned, while on the other hand, information that would be most useful to us in our effort to correlate experimental with clinical phenomena is not provided by them. For example, they make no very definite statement as to the degree of paralysis of voluntary movement in the crossed rigid limbs. It appears

that there is some weakness, but not absolute loss of power, and to what extent it is permanent we are not told. A further study of hemidecerebrate rigidity is therefore called for before a complete comparison with hemiplegia in the human subject is possible.

4. *The Physiologic Qualities of Decerebrate Rigidity.*—Not less important than the aspects of the question we have been considering are the qualities of the rigidity. In his paper on The Postural Activity of Muscle and Nerve in *Brain* in 1915, Sherrington gave a minute account of these.[10] Muscle tone, and hence decerebrate rigidity also, is a proprioceptive reflex arising in the tonic muscle. Hence posterior root section, or "de-afferentation," as Sherrington expresses it, abolishes tone in the muscle. The "plasticity" of the rigidity is the resultant of two distinct reflex reactions, the "shortening" and "lengthening" reactions, by which the muscle tends to retain a new length imposed on it either by active contraction or inhibition, or by passive manipulation. In any sustained movement, the intervals which would otherwise occur between the component contractions are bridged over, and a continuous, instead of an intermittent, contraction results. Like all tonic reflex muscular reactions, decerebrate rigidity is readily inhibited for the time being by reflex reactions of phasic type. Thus, flexion, crossed extension and extensor thrust reflexes are obtainable in the decerebrate preparation on appropriate stimulation. These reactions break through the tonic posture of the animal, which however is at once restored by a compensatory rebound as soon as the movement is completed. Another characteristic feature is the occurrence of clonus.[11]

5. *Other Forms of Muscular Spasm Producing an Extensor Attitude: Tonic Fits.*—We have seen that the tonic contraction of the extensor muscles in the decerebrate animal produces a general attitude of extension, but, in uncomplicated cases, without much head retraction or opisthotonus. We must now consider whether there is anything specific in this general attitude for the form of rigidity now under discussion, and whether it can be taken as an infallible indication of the presence of decerebrate rigidity.

A moment's thought will show that both these questions must be answered in the negative. This posture is familiar to us in the generalized tonic spasms produced by strychnin and tetanus toxin poisoning, both of which, as Sherrington was the first to point out,[12] are

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10. Sherrington, C. S.: Postural Activity of Muscle and Nerve, Brain **38**: 191, Pt. 3, 1915.

11. Viets, H.: Relation of the Form of the Knee Jerk and Patellar Clonus to Muscle Tonus, Brain **43**:269, Pt. 3, 1920.

12. Sherrington, C. S.: The Integrative Action of the Nervous System, London, Constable & Co., 1906; New York, Charles Scribner's Sons, 1906.

examples of generalized spasm of all the skeletal muscles, that is, of an overthrow of reciprocal innervation, in contradistinction to the selective extensor spasm of decerebrate rigidity. Further, not only may generalized muscular spasm produce the so-called decerebrate posture, but Sherrington [1] has observed that decerebrate rigidity is but a type of extensor spasm of which allied examples follow various other lesions of the cerebellocerebral region.

This brings us to the very important question of tonic fits. The circumstances under which these may occur in the experimental animal throw considerable light on their appearance in man, and indicate their essential difference from the reflex tonic contraction we have been considering. Bazett [13] working in Sherrington's laboratory, found that "the occurrence of tonic fits is comparatively rare, and is limited to the first few hours after the operation, except in some cats in which meningitis has developed. In the early cases the opisthotonus is extreme, the respiration is generally slow, but may be fast and shallow, and occasionally with a slow respiratory rate the fits themselves have a respiratory rhythm. These animals are very different from the better preparations in which the respiratory rate is normal, and they rarely survive long if the fits are at all severe. The operation often causes a certain amount of hemorrhage below the tentorium into the subarachnoid space, but this hemorrhage seems to have been particularly noticeable in the animals which have shown these fits." In Bazett's later paper with Penfield these remarks are amplified. They indicate clearly the process operative in the production of the fits. The reflux of blood round the medulla leads to compression of this structure and to interference with the blood supply of the medullary centers. The initial reaction of nervous tissue to oxygen starvation, or anoxemia, is the development of hyperexcitability. The fits and the respiratory irregularities are discharges from anoxemic centers.[14] Therefore, the fits are not reflex reactions, but are the equivalent of stimulation experiments.

Speaking of similar discharges from the cells of the spinal cord, and contrasting them with reflex motor reactions, Sherrington [15] says "In the same way, asphyxia, especially when rapidly produced, evokes discharges of the motor neurones of the cord, and the discharge produced by it, is of both extensors and flexors, and occurs as readily

13. Bazett, H. C.: Brain **43**:306, Pt. 3, 1920.

14. Walshe, F. M. R.: On the Physiology of Symptom Production in Disease and Injury of the Nervous System, Brit. M. J. **2**:837 (Nov. 19) 1921; The Rôle of Anoxaemia in the Production of Symptoms in Disease and Injury of the Nervous System, Med. Science **4**:427 (Aug.) 1921.

15. Sherrington, C. S.: Spinal Reflex Action, Croonian Lecture, 1897, Phil. Trans. Roy. Soc., B. **190**:128, 1898.

when all the afferent roots remain intact. The chemical excitation of the asphyxial condition so far resembles that of strychnia that in contradiction to the tonic conditions under discussion here, it is due to an excitation not conveyed via the afferent roots but must be autochthonous in the gray matter of the cord. . . . Both stand out together in strong contrast with such a condition as decerebrate rigidity . . . " These remarks may be applied to tonic fits.

In conclusion, not only is strychnin and tetanus spasm of totally different quality from decerebrate rigidity, but it appears highly probable that tonic fits are also generalized muscular spasms produced by a form of chemical excitation, and therefore equally distinct from decerebrate rigidity, which is a pure reflex reaction showing reciprocal innervation. Yet in every instance the general attitude of the animal is much the same, except that in generalized spasm head retraction and opisthotonus are more marked. Therefore, the term decerebrate posture is misleading and devoid of precision.

Finally, one further observation may appropriately be made here, namely, that neither Sherrington, Magnus, Bazett, nor any other authority has recorded the occurrence of involuntary movements of the tremor, choreiform or athetosis varieties in the decerebrate animal.

IV. CLINICAL CONSIDERATIONS

The conception of decerebrate rigidity as a condition occurring in man is not altogether new. However, it is only since the publication of Wilson's recent paper that the subject has aroused a general interest among clinical neurologists, and with the appearance of each new study of the question it is becoming more apparent that already clinicians understand something very different from the clearly defined condition described by Sherrington and named decerebrate rigidity.

1. *Spasticity in Hemiplegia and in Paraplegia in Extension as a Form of Decerebrate Rigidity.*—It was the study of the spasticity following lesions of the pyramidal system that first suggested to clinical neurologists that a form of decerebrate rigidity was not uncommon in man. The idea found its first general expression in 1901 in a paper by Collier and Buzzard on descending mesencephalic tracts in the cat, monkey and man.[16] In 1905, Thiele[2] opened the discussion of his experimental observations with a consideration of spasticity in man as a form of decerebrate rigidity, but he made no clinical studies to confirm his analogy. In the following year, in his Hughlings Jackson Lecture,[5] Horsley referred to decerebrate rigidity in this connection and correlated

16. Collier, J. S., and Buzzard, E. F.: Descending Mesencephalic Tracts in Cat, Monkey and Man, Brain **24**:177, Pt. 2, 1901.

Jackson's "cerebellar influx" with Sherrington's decerebrate rigidity. In 1914, and again in 1919, the present writer recorded a systematic analysis of various types of spastic paralysis in the light of Sherrington's experimental researches on the nervous system.[17] It was pointed out that in a spastic hemiplegia, and in paraplegia in extension the lower limbs lie fully extended and adducted with the feet plantarflexed (i. e. extended). The limb extensors (including plantarflexors) are in a state of increased tone, give brisk tendon reflexes and may show clonus. The antagonistic flexor (and dorsiflexor) groups, on the other hand, not only retain much less voluntary power than the extensor group, but show a peculiar phasic type of reflex activity, which is seen in the flexion reflex with its component Babinski plantar response. This group does not present the characteristic spasticity of the extensors. The physiologic differentiation of the two groups was discussed, and it was concluded that in the hypertonus of the extensors we had a form of tonic muscular activity identical with experimentally produced decerebrate rigidity, and like this possessing a long reflex arc extending into the brain stem. An analysis of spasticity confirmed this view. The incidence of the spasticity is selective in the limb extensors. It shows both shortening and lengthening reactions; the former seen in the tonic prolongation of the knee jerk under these conditions, and the latter seen as the clasp-knife quality of the rigidity on passively lengthening a muscle, as, for example, on flexing the knee.

Further, de-afferentation of the tonic muscle by posterior root section abolishes the rigidity. Finally, just as decerebrate rigidity in the animal preparation can be temporarily overset (reciprocally inhibited) by such phasic reflex reactions as the flexion and crossed extension reflexes, so in man, these two reactions were identified and found to cause a temporary modification of extensor spasticity. Graphic records were given demonstrating several of these points.[17] In the case of the lower limb, therefore, no single physiologic quality was wanting for the complete identification of spasticity with experimentally produced decerebrate rigidity. In the case of the upper limb, however, a special problem was presented, for this commonly becomes spastic in flexion. It has been pointed out by several writers that the upper limb in man has lost its primitive function as a locomotor prop and has taken on new and highly complex activities, in which flexion rather than extension may be said to be the predominant factor. Possibly,

17. Walshe, F. M. R.: The Physiological Significance of the Reflex Phenomena in Spastic Paralysis of the Lower Limbs, Brain **37**:269, Pt. 2, 1914; On the Genesis and Physiological Significance of Spasticity and Other Disorders of Motor Innervation, with Special Reference to the Functional Relationships of the Pyramidal System, Brain **40**:1, Pt. 1, 1919.

the profoundly altered physiologic rôle of this limb may contain the clue to the peculiar incidence of spasticity in the flexors. Be this as it may, the studies of Riddoch and Buzzard [18] on the reflex movements of the arm indicate that here, as in the lower limb, phasic reflex reactions can temporarily overset the hypertonus, and there can be little doubt but that in quality the rigidity in the two limbs is identical.

It is clear, however, that we can scarcely speak of the patient with hemiplegia, still less the one with paraplegia in extension, as wholly decerebrate. Further, there are considerable difficulties as soon as we attempt to correlate the lesions producing these states of the musculature in man with those essential to the production of decerebrate rigidity in animals. We cannot hope to see reproduced in man the complete anatomic transections possible in animals. It has generally been assumed that a lesion of the pyramidal system is an adequate cause not only of loss of voluntary power, but also, through release of subordinate motor mechanisms, of spasticity. It seems probable that in respect of the latter we shall have to modify our views. The occurrence of paralysis without spasticity as a result of lesions confined to the excitable motor cortex has long been known.[19] Moreover, Collier and Buzzard in the paper already referred to, and later Marie and Guillain [20] have observed that pyramidal degenerations are more bulky when the lesion is situated in the subthalamic region or caudad than when it is in the forebrain. Therefore, what we speak of as a pyramidal tract lesion must frequently involve non-pyramidal fibers in addition. There are also some indications that lesions of the frontopontine fibers may play a part in the genesis of spasticity. In short, in man, as in the experimental animal, there are at least two factors essential to the production of that spasticity which we believe to be physiologically identical with decerebrate rigidity, namely, interruption of the corticospinal path at some part of its course, and interference with the functions of the brain stem mechanisms concerned in the maintenance and regulation of postural tone, or of afferent or efferent paths in relation with them. The occurrence of spasticity in spinal diseases indicates that lesions of descending paths from the brain stem centers may suffice to produce an effect similar to that of a lesion in the centers themselves. We must admit, however, that the

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18. Riddoch, George, and Buzzard, E. F.: Reflex Movements and Postural Reflex Reactions in Quadriplegia and Hemiplegia, with Especial Reference to Those of the Upper Limb, Brain **44**:397, Pt. 4, 1921.

19. Bergmark, G.: Cerebral Monoplegia, with Special Reference to Sensation and to Spastic Phenomena, Brain **32**:342, 1909.

20. Marie and Guillain: Les dégénérations secondaires du cordon anterieur de la moëlle, Rev. Neurol., 1904, No. 14.

anatomic correlation of the clinical with the experimental problem is not yet complete, notwithstanding that the physiologic study of spasticity provides ample evidence of the identity of the two.

2. *"Decerebrate Man" and Tonic Fits.*—In 1912, Magnus and de Kleijn described a series of five cases of what they believed to be an approximation to the experimental condition of decerebration, in that there was a more or less complete abolition of cerebral function in all.[8] Clinically, however, except that in all the cases various Magnus and de Kleijn postural reflexes were observed, they form a very heterogeneous series. Thus, Case 1 was that of a child with hydrocephalus and optic atrophy. The child was unconscious and wholly atonic throughout the months of observation. Case 2 was that of a child with hydrocephalus, who walked with a spastic, ataxic and paretic gait, and in whom there cannot have been a very profound abrogation of cerebral function. Case 3 was an old man, the subject of a ventricular hemorrhage. He survived this for less than forty-eight hours, during which period he lay in rigid extension with arms extended and adducted. Tonic fits are not mentioned. It is probable that his rigidity was not reflex in origin, but the result of increasing medullary compression and of direct stimulation of brain stem centers by asphyxial blood. Case 4 was that of a new-born child, which did not breathe for nearly an hour after a difficult delivery. It was in strong extensor rigidity with tremor. This appears to have lasted less than a week, and for the remaining three months of life there was no rigidity. Case 5 was one of septic meningitis with convulsions.

The cases of their second series[21] in 1915 are equally mixed, and both series may be taken to show, not so much the presence of decerebrate rigidity in man, as the appearance of tonic labyrinthine and neck reflexes after various cerebral lesions. Indeed, it was the demonstration of these reflexes that Magnus and de Kleijn were primarily concerned to carry out. Brouwer,[22] Winkler, Weiland and De Bruin, all Dutch neurologists, have from time to time published isolated cases of the same kind, and like Magnus and de Kleijn have been more concerned to demonstrate tonic neck and labyrinthine reflexes than to

21. Magnus, R., and De Kleijn, A.: Weitere Beobachtungen über Hals- und Labyrinthreflexe auf die Gliedermuskeln der Menschen, Arch. f. d. ges. Physiol. **160**:429, 1915; Kleinhirn, Hirnstamm und Labyrinthreflexe, München. med. Wchnschr. **66**:523, 1919; Ueber die Unabhängigkeit der Labyrinthreflexe vom Kleinhirn und über die Lage der Zentren für die Labyrinthreflexe im Hirnstamm, Arch. f. d. ges. Physiol. **178**:124, 1920.

22. Brouwer, B.: Klinisch-anatomische Untersuchung über partielle Anencephalie, Ztschr. f. d. ges. Neurol. u. Psychiat. **32**:164, 1916; Ueber Meningoencephalitis und die Magnus-de-Kleijnschen Reflexe, ibid. **36**:161, 1917.

verify by the necessary investigation the presence of true decerebrate rigidity in their subjects. None of these writers has made any attempt to study the quality of the rigidity in such of their cases as it was present.

In 1920, Wilson [23] gave a fresh impetus to the clinical study of the subject, but, unlike the authors referred to above, he was primarily concerned with the recognition of decerebrate rigidity in man, and gave very little attention to Magnus and de Kleijn's tonic reflexes. Of his cases it may be said that they include every clinical variety of disorder of motor function; spastic paralysis, extrapyramidal syndromes, and cerebellar symptoms, while tetanus and hysterical spasm are also quoted as manifestations of decerebrate rigidity. Throughout his paper the attitude of extension, either general or confined to a limb or limb segment, and with or without muscular spasm, is taken as the sole criterion by which the identification with experimentally produced decerebrate rigidity is established. No attempt is made to exclude generalized incoordinate muscular spasm, nor to distinguish manifestations of "release of function" from those of direct excitation or stimulation. Finally, the quality of the muscular spasm present has not been studied. In these circumstances, it can only be by chance that genuine examples find inclusion in so heterogeneous a collection of clinical conditions. From the brief data provided by Wilson it seems highly probable that his Cases 6 and 9 are examples of decerebrate rigidity, but for the rest there is no obvious basis for the diagnosis, except the fallacious one of attitude. It is not necessary to add to the remarks already made on this point, and Wilson's use of the words "attitude" and "rigidity" as synonymous and his statement that "tonic fits are in reality attacks of decerebrate rigidity" reveal how wholly different from the physiologist's is his conception of decerebrate rigidity.

In this connection we note that the tonic fits described and quoted by Wilson all occur in the same circumstances of gross disturbance in the blood supply to the brain stem as obtained in the case of Bazett and Penfield's cats, namely, pressure by tumors or by hemorrhage. It is in strict accord with this that the signs of direct stimulation, or to use a less accurate but more familiar term "irritation," are present in all. These are gross irregularity in force and frequency of respiration, bradycardia, hyperpyrexia, cyanosis, fixed dilated pupils, with extreme head retraction and opisthotonus. All of these terminate more or less rapidly in death, and are no part of reflex decerebrate rigidity.

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23. Wilson, S. A. K.: On Decerebrate Rigidity in Man and the Occurrence of Tonic Fits, Brain **43**:220, Pt. 3, 1920.

In short, a survey of the clinical literature indicates that the term decerebrate rigidity has been applied in man to conditions of three main types (1) Spastic paralysis: Here the chief justification is the fact that, physiologically considered, spasticity is identical with experimentally produced decerebrate rigidity. (2) Tonic muscular spasm in extension in comatose patients, with or without tonic fits. The examples so far recorded in this group include various forms of generalized tonic muscular spasm due to direct excitation of grey matter, and in none of them have the essential qualities of the rigidity been investigated or verified. Further, a fallacious standard, that of attitude, has been adopted as the sole and essential one. (3) Cases of so-called fragmentary decerebrate attitude. By no adequate standard can these, as recorded hitherto, be accepted as in any way related to the experimentally produced decerebrate rigidity.

3. *The Clinical Application of the Facts of Experimental Observation.*—As a preliminary to the study of some illustrative cases, we may now summarize the characteristics by which we may hope either to establish or to reject a diagnosis of decerebrate rigidity in man.

1. The Quality of the Rigidity.—This will be of the kind familiar to us in spastic hemiplegia and in paraplegia in extension, the so-called spasticity of pyramidal system lesions. It is a true hypertonus, employing Sherrington's sense of the word tonus. It is therefore a proprioceptive reflex reaction, selective in its incidence in the musculature and showing reciprocal innervation. It is abolished by posterior root section. It presents the quality of plasticity described by Sherrington, though, in longstanding cases of spastic paralysis, fibrous contracture tends to mask this. Clinically, the two component reactions which confer plasticity, the shortening and the lengthening reactions, are readily observed, the former as the tonic prolongation of the tendon jerks, and the latter as the sudden yielding of the rigidity on passive stretching of the muscle, with the adoption of a new and increased length, the clasp-knife rigidity of clinical writers. Like all tonic reactions, spasticity is readily overset or inhibited by certain phasic spinal reflexes, such as the flexion reflex. This reflex, with its component the Babinski plantar response, is readily demonstrable in man as in the decerebrate animal. Clonus, another characteristic reaction of tonic muscles, is also found in man in these circumstances.

In man, however, in the case of the upper limb, spasticity in flexion is more common than in extension, but as Riddoch and Buzzard [18] have shown the latter form does occur, and in both instances they have found that phasic spinal reflexes may momentarily inhibit and replace the rigidity, as in the decerebrate animal.

2. Clinical Types.—From what has been said it is clear that muscular rigidity comparable with experimentally produced decerebrate rigidity is most commonly seen in man in association with hemiplegia, with cerebral diplegia of pure pyramidal type and in paraplegia in extension from spinal cord lesions. It is possible, also, that from time to time, there occurs in man a more exact approximation to the condition of the decerebrate animal, with bilateral extensor rigidity and total abrogation of cerebral function. However, such a condition has not yet been satisfacforily established by clinical observation. Moreover, it is highly probable that the cases hitherto put forward as examples of decerebrate man have been instances of generalized muscular spasm resulting from direct stimulation of grey matter in pons and medulla by compression and a resulting anoxemia. The rôle of anoxemia in this connection has been fully discussed by the present writer elsewhere.[14] Strychnin and tetanus spasm tonic fits are examples of direct stimulation and not of true reflex muscular spasm.

Therefore, considerable caution is necessary in accepting as cases of decerebrate rigidity in man, all those which present so-called irritative symptoms such as cardiac and respiratory irregularities, especially when these lead rapidly to respiratory failure and death, cyanosis, a rising temperature, fixed and dilated pupils and tonic fits.

3. Extrapyramidal Motor Disease.—It follows from these considerations that all forms of extrapyramidal motor disease associated with muscular rigidity, with or without involuntary movements, must be rejected as having no relation with decerebrate rigidity. In such cases, the qualities of the rigidity and its incidence, and the absence of the essential interference with the functions of the pyramidal system clearly indicate that we are dealing with a fundamentally different set of phenomena.

4. Attitude of Extension.—It has to be remembered that an attitude of extension of head, trunk and limbs is not peculiar to decerebrate rigidity, and its presence cannot be regarded as diagnostic in the absence of the other and specific features we have mentioned.

In the light of Magnus and de Kleijn's work on postural reactions, we shall in future have to look for the presence of these reactions in cases of suspected decerebrate rigidity in man. Apart from the studies of the Dutch neurologists referred to earlier, no systematic investigation on these lines has yet been recorded.

Finally, it may not be superfluous to emphasize that decerebrate rigidity, as described by Sherrington, is a very clearly defined condition. If we are to adopt this term of precision from experimental physiology we must be careful to use it precisely, and we have no right to employ it to describe clinical conditions which differ in essential features from the experimentally produced one known by that name.

REPORT OF CASES

In this section there are described three cases: one of residual hemiplegia with spasticity; one of tonic fits without intervening rigidity; and one of generalized muscular rigidity of extrapyramidal type. They are analyzed in the light of the physiologic conception of decerebrate rigidity, and reasons are given for concluding that the first alone is an example of this condition, while the second and third illustrate two other and fundamentally different forms of tonic muscular spasm.

CASE 1. *Cerebral capsular thrombosis with residual spastic hemiplegia of the ordinary type. This spasticity presents the qualities of decerebrate rigidity.*

History.—A. C., a man aged 45, fell suddenly unconscious from a chair in April, 1913. He states that he was unconscious for over a day, and that on recovery he found himself totally paralyzed down the right side and with great difficulty in articulation. He was admitted to the National Hospital, Queen Square, under the care of Dr. James Collier within a month of the onset. His condition at that time was one of right hemiplegia with dysarthria and slight right hemianesthesia. The right arm was spastic in flexion, and except for feeble elevation at the shoulder no voluntary movement was possible in the limb. The leg was spastic in extension and had recovered sufficient power to render walking with support possible. The reflex changes were those usual in the condition. The viscera were healthy. The Wassermann reaction in the blood was negative.

Examination in December, 1922, revealed a residual hemiplegia, no defect of articulation and no objective sensory loss. There was slight spontaneous aching pain in the affected arm and leg and a definite over-response to cutaneous stimuli of nocuous character on that half of the body.

Motor System.—As the patient stood, there was a visible paresis of the lower part of the face on the right; the right arm was adducted, flexed and held across the body; the right leg was extended and the foot pointed. The motor cranial nerves were normal, with the exception of the right facial paresis. The right upper limb had regained power in coarse movements at the shoulder, elbow and wrist. At the two latter joints the power of flexion was distinctly better than that of extension. Full range of extension was impossible. He could not extend the clasped hand, but when the observer's finger was inserted into the fist, he was able to clench with moderate power. On all forced movements of the opposite (normal) limb, there was involuntary associated flexion of the paralyzed arm at all joints. There was considerable spasticity in the flexors of elbow, wrist and digits and in the adductors of the shoulder, but there was no permanent contracture. Once the limb was passively extended, no spasticity could be demonstrated in the extensors and passive flexion met with no tonic resistance.

The right lower limb was extended, adducted and the foot plantarflexed. The hallux was in moderate dorsiflexion. Voluntary extension and plantarflexion were of good, though not normal, force. Flexion at hip and knee was very feeble, while there was no voluntary power of dorsiflexion of the foot, and very little of the digits. There was considerable spasticity in the hip and knee extensors and in the plantarflexors of the foot.

Tendon Reflexes.—The tendon reflexes were brisk on both sides, but more markedly so on the right, where there was both patellar and ankle clonus. The right abdominal reflexes were absent, those of the left present. The right plantar response was extensor in type. It was obtainable from a cutaneous receptive field including the sole and dorsum of the foot, and the leg up to the knee and from the skin on the mesial aspect of the thigh in its lower half. It could also be obtained by pressure on muscles, tendons and bones up to the level of the knee. When strong stimuli were used it was invariably accompanied by visible flexion of the limb at knee and hip. When the sole was lightly stroked, no visible flexion was seen, but there was palpable and visible contraction in the hamstring muscles. In other words, a fully developed nociceptive flexion with its component Babinski response was present, and could be elicited from a wide receptive field. There was no crossed extensor reflex (that is, crossed plantarflexor hallux reflex).

The case presents the type of spasticity characteristic of residual hemiplegia. It was selective in incidence, being found in the flexors of the upper and the extensors (and plantarflexors) of the lower limb. Clonus was present. The spasticity was, in the case of the lower limb, readily overset by the phasic spinal flexion reflex which was obtainable from a wide cutaneous and deep

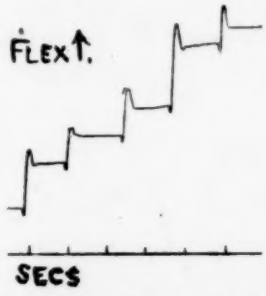


Fig. 2.—A series of supinator jerks showing tonic after-contraction, or shortening reactions. The vertical movement is the record of each tendon reflex. Relaxation is seen to be very incomplete, so that an additional degree of shortening of the muscle remains after each reflex. Taken on a slow moving kymograph drum. Time is recorded in seconds. The amplitude of the record is one-fifth that of the actual excursion.

receptive field. In the case of the upper limb, the phasic reflex described by Riddoch and Buzzard was not obtained. The quality known as plasticity was also present and was readily demonstrated in the upper limb. As the patient stood with flexed arm, the forearm was pronated and held horizontally. In this position the supinator jerk was readily obtained without supporting the limb. Thus elicited, the reflex was brisk and ample, but relaxation was not complete and after each of a series of rhythmically repeated taps on the lower end of the radius, the degree of flexion of the forearm on the upper arm was slightly increased. This is well seen in the accompanying records, Figs. 2 and 3.

In Figure 2, taken on a slow moving kymograph drum, after each tap on the tendon there is seen a rapid twitch with incomplete relaxation, so that the subsequent reflex starts from a higher level, and a step-ladder record is produced. In Figure 3, taken on a fast moving drum a similar tonic after-

contraction, or shortening reflex, is also seen and can be contrasted with a similar record taken from the normal arm, where this is not present. The reciprocal inhibition of the spasticity which occurs when a flexion reflex is elicited is also capable of graphic record. The patient was laid on his back and the patellar tendon and hallux were connected with the kymograph. While an assistant pressed the patella distalwards and produced a patellar clonus, the sole of the foot was stroked and a flexion reflex produced. Flexion at hip and knee was prevented by fixing the thigh and leg to the board on which they lay extended. It is seen (Fig. 4, *A, B*) that the clonus and the length of the vasti muscles are materially modified by the intercurrent flexion reflex. In Figure 4, *A*, the clonus is completely inhibited and there is a

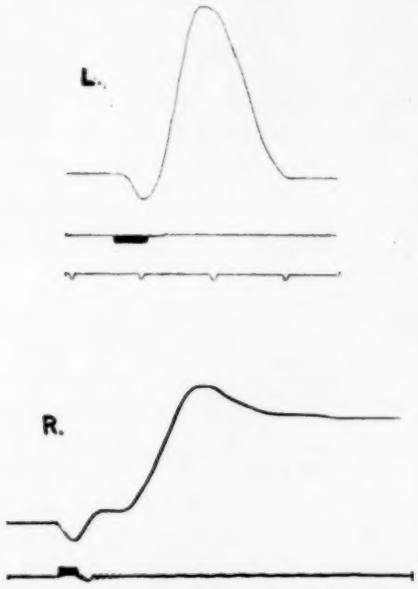


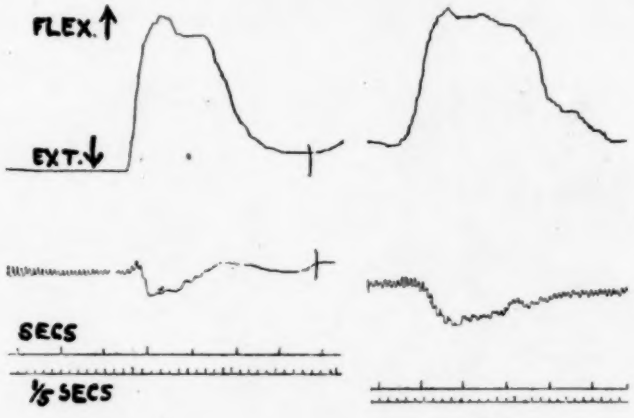
Fig. 3.—Supinator jerks from same patient taken on a rapidly moving drum. The upper record is from the normal arm (L) and shows complete and rapid relaxation of the muscle, the lower is from the spastic arm (right) and shows the shortening reaction. The initial downward movement in each record is due to the depression of the forearm caused by the tap on the tendon and is seen to be synchronous wih the signal, which indicates the time and duration of the tap. The upper record is twice the amplitude of the actual excursion of the limb and the lower only three-fourths that of the actual reflex. These modifications were introduced to render the two records approximately equal in range, and do not affect either the form or time relations of the two jerks. For technical details see appendix.

distinct though slight lengthening of the vastus. In B, the clonus is not completely extinguished, but diminished in amplitude and the same tonic lengthening of vastus is seen.

Postural reflexes (tonic labyrinth and neck reflexes of Magnus and de Kleijn).—Distinct evidence of these reactions was present. Neck reflex: the

patient was laid on his back and the head rotated away from the right (paralyzed) side, that is, with the face pointing to the left shoulder. The head was then slowly rotated by the observer to the opposite side, that is, with the face pointing to the right shoulder. As rotation became complete a slow extensor spasm occurred in the flexed right arm, which became almost fully extended at the elbow, while the forearm went into full pronation. The head was now rotated back again to its original position, that is, face pointing to left shoulder. As this movement was being completed, the extensor spasm relaxed and the arm returned to its initial position of flexion. No similar response was obtained from the lower limb in this case.

Vestibular reflex: Magnus found that the reinforcing influence of the labyrinths on extensor tonus was maximal when the animal lay on its back with the snout pointing upwards. In this patient, no variation of limb tonus was found on laying him down from a sitting position. However, signs of



A B

Fig. 4 *A* and 4 *B*.—Showing reciprocal inhibition of patellar clonus, which is a phenomenon of the extensor group, by the flexion reflex, a reaction employing the antagonistic flexor group. The upper record is that of hallux and foot dorsiflexion. Hip and knee flexion were prevented by fixing the limb. Therefore, the upper record comprises the Babinski plantar response plus dorsiflexion of the foot. The record is one-third the amplitude of the actual excursion. The lower record is that of the clonus. Upward movement represents contraction of the vasti and downward movement lengthening. In *A*, the clonus is abolished and there is a lengthening reaction of vasti. In *B*, the clonus is not completely abolished, but goes on at a greater length of the reacting muscles. Time is recorded in seconds and in fifths.

labyrinthine influence were observed in that the extensor spasm in the upper limb produced by rotation of the head (face to right shoulder) was more ample in range and more forceful when the patient lay on his back than when he was standing. Technical difficulties excluded the graphic recording of this labyrinthine reaction.

Comment.—The case is one of residual hemiplegia with spasticity. It is seen that the spasticity is selective in incidence and presents: clonus; reciprocal

inhibition by the phasic spinal flexion reflex; the shortening reaction; and the presence of the postural reflexes of Magnus and de Kleijn. In short, the spasticity presents all the features of experimentally produced decerebrate rigidity.

CASE 2. *Partial anencephaly, total absence of forebrain and of part of midbrain. Survival after birth, eighteen hours. No rigidity, but tonic fits produced at will by pressure on defect in skull. These fits were found to differ essentially from true decerebrate rigidity.*

History.—M. B., a female child, delivered at full term, weighed 7 pounds, and was of normal development except in respect of the skull and brain. This monster will be the subject of later detailed anatomic and clinical reports, and is dealt with here simply as demonstrating the probable nature of tonic fits.

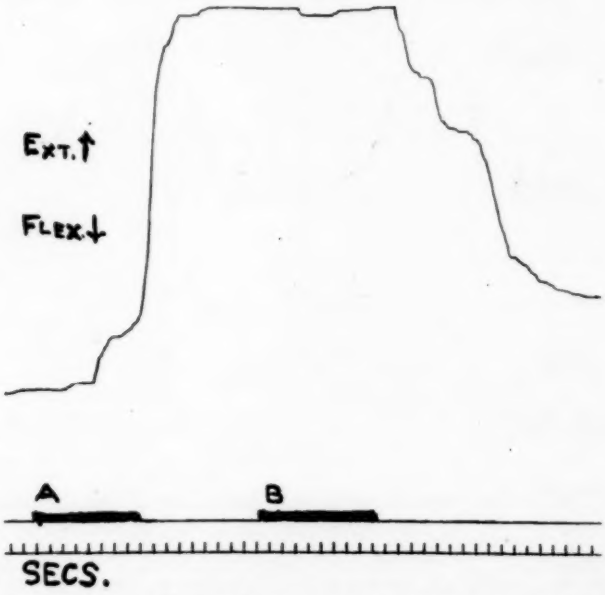


Fig. 5.—Tonic neck reflex (Magnus and de Kleijn) in right arm. Here, upward movement indicates extension of the limb, and downward movement flexion. Of the two records below, the upper at *A* records the time and duration of head rotation to the right (face points to right side), and *B* time and duration of head rotation back to original position (face pointing to left side). It is seen that as the initial rotation is becoming complete the right (paralyzed) arm goes into strong extensor spasm. This relaxes when the rotation of the head back again to the left is nearly complete. The record is of one-fifth the amplitude of the actual excursion of the limb. Extension was also accompanied by complete pronation of the forearm. Time in seconds.

The child was examined twelve hours after delivery. There was no external abnormality except in the case of the head. The whole of the calvarium seemed to be absent. There was no skull or scalp above a line drawn round the head immediately above the eyes in front, above the pinnae lat-

erally and thence carried round the occipital region posteriorly. There was a fringe of hairy scalp above the ears and posteriorly. The position of the cerebral hemispheres was occupied by a small, soft, pink, lobular mass, which in no way resembled cerebral tissue to naked eye inspection, and did not pulsate. The head was retracted, the eyelids were extremely swollen and approximated and the eyes were not seen.

The child lay immobile in the normal attitude of the newborn. There was neither extensor attitude nor extensor rigidity. There was clearly no temperature regulation and the temperature gradually sank from the time of delivery. At the moment of examination it was 96 F., and the child felt cold. Respirations were 32 per minute, markedly irregular in force and frequency. The child could suck, and cried when handled. It was cyanosed. The abdominal reflexes were brisk and equal on the two sides, the knee jerks were present, but the ankle jerks were not obtainable. The plantar responses were either simple plantarflexion, or plantarflexion followed immediately by dorsiflexion of the hallux and some limb flexion. The flexion was most pronounced

Fig. 6.—Drawings of head of monster (Case 2), for which I am indebted to Miss Ruth Clulow, house physician to the medical unit. The shaded areas indicate the soft tissue replacing the cerebral hemispheres. The attitude of the head in the profile drawing is that taken up during a tonic fit. The swollen and closed eyelids are readily seen.

on pricking the sole with a pin, and in these circumstances there was, in addition, an elevation of both arms at the shoulders. No Magnus and de Kleijn reflexes, labyrinthine or from the neck, were obtainable. In this respect the case agrees with a similar one recorded by Brouwer.[20]

Transient pressure on the pink mass presenting on top of the head produced rapid flexion movements of the limbs, sometimes unilateral, but generally bilateral. A tonic fit could be produced by pressure with the pulp of the thumb on the center of the pink mass and maintained for two or three seconds. At the expiration of this time, the child went into opisthotonus, with legs rigidly extended and adducted, feet plantarflexed, and the arms adducted and flexed at the elbows with the fists clenched and the forearms half pronated. Respiration ceased entirely, the chest became fixed in strong tonic spasm and there was deep and increasing cyanosis. Examination of the musculature

now revealed intense tonic spasm in all muscles, every muscle was hard and contracted. No reflex responses could be obtained, either tendon jerks, abdominal reflexes or plantar responses. The condition was one of intense tonic spasm of the entire skeletal musculature, and clearly distinguishable from true reflex decerebrate rigidity. Each fit lasted for from forty to fifty seconds and then suddenly passed off. Respiration was resumed and the cyanosis diminished in intensity, but never entirely disappeared. Such fits could be produced in the manner described at short intervals and without fail.

Anatomic investigation of the nervous and other systems of this monster is at present being carried out in the Department of Anatomy, University College (Professor Elliot Smith), by Dr. H. A. Harris, who will in due course publish a full anatomic account of the specimen. In the meantime, and for our present purpose, which is simply the description and analysis of a tonic fit, it suffices to say that the whole forebrain and 'tween brain and the cephalad portion of the midbrain were absent. Therefore, anatomically the monster was a decerebrate animal, but showed no trace of decerebrate rigidity, nor of any form of muscular spasm in the intervals between the fits described.

Comment.—A full-term child born without forebrain and surviving delivery for several hours. It displayed no persistent decerebrate rigidity and its attitude was that normal for the new-born child, except during the tonic fits described. Temperature regulation was absent, respiration was irregular in force and frequency and there was cyanosis throughout the survival period. The reflexes present have been described. In these circumstances, pressure upon the unprotected brain stem evoked tonic fits of the type described by Hughlings Jackson and later writers, and differed from certain of them in the involvement of the respiratory musculature in strong tonic spasm and temporary suppression of the respiratory movements. The tonic muscular spasm was generalized and differed in this respect, and in the reflex reactions obtainable during its presence, from true reflex decerebrate rigidity. The mechanism of its production appears to have been the common one, namely, a sudden increase in the degree of asphyxia present throughout survival, which sufficed to render the gray matter of the brain stem hyperexcitable and to cause a discharge into all the motor neurons of the skeletal musculatory.

CASE 3. *Generalized muscular rigidity following an attack of lethargic encephalitis and of eighteen months' standing. No true paralysis, but movements prevented by intensity of muscular spasm. No signs of pyramidal lesion. The condition not related to decerebrate rigidity, although the general attitude is one of extension.*

History.—A. F., a carman, aged 36, was admitted to the National Hospital under the care of Dr. Aldren Turner in December, 1922, with the following history. He was originally admitted to Winchester Hospital in January, 1920, with an acute illness diagnosed as lethargic encephalitis. There were no focal nervous symptoms recorded except a diminution of the knee jerks, and radiating pains in the legs. In January, 1921, he was noted to have rigidity of the jaw muscles which prevented him from freely opening his mouth, absence of the knee jerks and general weakness. By November, 1921, he had developed a generalized muscular rigidity and looked and held himself like a case of paralysis agitans without tremor. During the ensuing year these symptoms increased in severity and for several months he had been bedridden and helpless.

Examination.—Dec. 12, 1922, he is seen to lie absolutely motionless in bed with his eyes closed, his under lip drawn in between his teeth, and his head, trunk and limbs extended. An exception must be made in the case of the right arm which is constantly flexed at the elbow and laid across the trunk. The muscular rigidity is intense and when moved or rolled about in the bed he is completely statuesque. He understands spoken speech and, when his eyes are opened, recognizes print and objects. He cannot articulate, but groans when he wishes to attract attention and can manage to indicate his needs in this fashion. Vision (acuity, fields and fundi) is normal. Hearing is normal. The pupils are equal, central and circular, but react very poorly both to light and to accommodation. There is an external squint of the right eye and convergence is defective, but lateral and vertical movements are normal. The face muscles, masseters and temporals are in strong tonic spasm. He cannot voluntarily open his eyes, except imperfectly and very slowly, but when they have been opened for him he can close them more rapidly and a little tremulously. He cannot make any facial movements, and there is absolutely

Fig. 7.—The patient A. F. (Case 3). At the time this photograph was taken the leg was slightly flexed at hip and knee, but this position was not invariable. When the pillow was taken from beneath the patient's head, this remained in space and did not fall back on the bed.

no play of expression over his features. The lower lip is constantly sucked in between the teeth. He cannot open his mouth, and the teeth are almost approximated, necessitating liquid feeds through a tube. He can swallow slowly and his feeding takes considerable time on this account. He cannot articulate. Painful stimuli to the skin of the face evoke no trace of reflex movement of the face muscles. The tongue has never been seen, nor have the palate and fauces, for it is not possible to open his mouth.

The Motor System.—The patient lies fully extended on his back with the neck extended, the lower limbs extended and adducted, and the feet fully plantarflexed. At times the knees are found crossed and slightly flexed (Fig. 7). The right arm is flexed at the elbow, adducted at the shoulder, with the forearm half pronated. The wrist is slightly flexed, and the fingers fully so and pressed into the palm. The extended thumb lies pressed against the index. The left arm lies fully extended and adducted. The forearm is fully pronated

and the attitude of the hand and digits is as on the right. All the muscles, neck, trunk and limb, are the seat of an intense rigidity, which persists undiminished throughout the whole range of passive movement at a limb joint. Flexing the head on the neck, or flexing the left arm at the elbow is like bending a lead pipe, and the new posture thus imposed is retained. In this sense the rigidity is plastic, but it differs from the clasp-knife character of pyramidal spasticity, in that the resistance to stretching does not abate throughout the whole range of movement. Further, in the case of the legs, manipulation in this way calls forth none of the reflex flexion spasms or clonus seen in spastic paralysis under these conditions. In the case of several joints, such as the shoulder, wrist, ankle and distal joints, a full range of movement is impossible with ordinary degrees of force. Further, in the limbs and trunk, each muscle belly stands out as though in strong voluntary contraction and is tense to palpation. Voluntary movement is almost extinguished by this spasm. Very slowly, and apparently with great effort, the patient can raise the arm to the vertical, or lift the extended leg from the bed. We may adapt a phrase in use among athletes and describe the patient as muscle-bound.

Reflexes.—The arm jerks are absent. The knee jerks are of normal briskness and rapidity. In this they are in strong contrast with the extreme slowness of voluntary muscular contraction. The intense plantarflexor spasm prevents the ankle jerks from being obtained. The abdominal reflexes are brisk and, like the knee jerks, rapid in contraction and relaxation. Stimulation of the soles evokes a normal plantarflexor response on the left and a variable response on the right. In neither case is there any associated hamstring contraction, but a brisk and rapid contraction of tensor fasciae femoris is visible. Apart from this, pin pricks, pinching of skin and muscles, and pressure on bones and tendons in the upper or lower limbs evoke no trace of reflex movement.

There is no sensory loss to such simple tests as are possible. There is normal sphincter control.

Comment.—There is an intense generalized tonic spasm of the whole skeletal musculature, which has imposed upon the patient an attitude of extension of head, trunk and limbs, with the single exception of the right arm which is flexed. The forearms are pronated and the digits clinched. In the case of certain muscles, permanent fibrosis has occurred and a full range of passive movement is not possible at all joints. This rigidity differs from experimentally produced decerebrate rigidity, not alone in having a diffuse incidence throughout the skeletal musculature, but also qualitatively. Inspection and palpation reveal firmly contracted muscles and a tendency to fixation in new attitudes passively imposed, of a kind not seen in decerebrate rigidity. With the single exception of the plantar responses, which are of normal type, there are to be seen no phasic spinal reflex movements on cutaneous and deep stimuli of nocuous character, such as are invariably associated with decerebrate rigidity in animals and with spasticity in man. Further, the increased amplitude of tendon jerks and their tonic prolongation, which are also an invariable accompaniment of decerebrate rigidity and spasticity, are absent. There is also no true paralysis, and in a word none of the signs which accompany what we regard as a lesion of the pyramidal system, an essential factor in the production of decerebrate rigidity in animals and of spasticity in man, are present.

The sole feature in this case capable of correlation with decerebrate rigidity is the attitude of the patient, and this, as we have seen, is not a feature peculiar to the condition in question. In short, the patient presents what has somewhat equivocally been called a decerebrate attitude associated with muscular spasm, and yet shows none of the characters of true decerebrate rigidity.

SUMMARY AND CONCLUSIONS

1. The anatomic factors essential to the experimental production of decerebrate rigidity are briefly stated, and the physiologic qualities and significance of this form of tonic reflex reaction described. The occurrence of other forms of muscular spasm, superficially resembling, but fundamentally differing from, true reflex decerebrate rigidity is discussed. The spasm of strychnin and tetanus toxin poisoning and tonic fits are identified as such forms of muscular spasm, unrelated to decerebrate rigidity.

2. The history of decerebrate rigidity as a clinical conception is briefly reviewed, and it is put forward that the spasticity of pyramidal system lesions was the form of tonic muscular contraction in man first regarded as akin to experimentally produced decerebrate rigidity, and the only form in which the identity has been systematically investigated and verified. The qualities of spasticity in man and of experimentally produced decerebrate rigidity are fully compared, and certain anatomic difficulties in the comparison are noted. It is suggested that many cases recently described as decerebrate rigidity in man are in all probability examples of those forms of generalized muscular spasm which are not related to reflex decerebrate rigidity, and further that they are for the most part manifestations of direct chemical stimulation of grey matter by defective oxygenation, or anoxemia, or forms of so-called extrapyramidal rigidity.

3. Finally, the standards to be observed in endeavoring to recognize decerebrate rigidity, as a clinically occurring condition in man, are stated, and three cases of different forms of tonic muscular spasm observed in man are reported. From the study of these, reasons are given for the conclusion that the spasticity seen in hemiplegia is physiologically identical with experimentally produced decerebrate rigidity, and that tonic fits and extrapyramidal muscular rigidity differ in essentials and are not related to it.

APPENDIX

The method of graphic recording employed in the analysis of Case 1 is indicated in the accompanying diagram (Fig. 8). The instrument consists of a standard Sherrington-Starling drum mounted on a base fitted with two special arms, the one having two light grooved pulleys

round which runs a thread carrying a writing point, and terminating in a long spiral spring at one end, and at the other being fastened to one of the five speeds of a large coned vulcanite pulley. From the latter another thread is led off to the limb or group of muscles under investigation. The excursion of the writing point may be varied in amplitude as required according to the grooves selected on the vulcanite pulley, on which are clips for securing the threads. The length of the thread leading to the subject can be adjusted in the well-known manner adopted to tighten tent ropes, and the tension of the spring can be varied

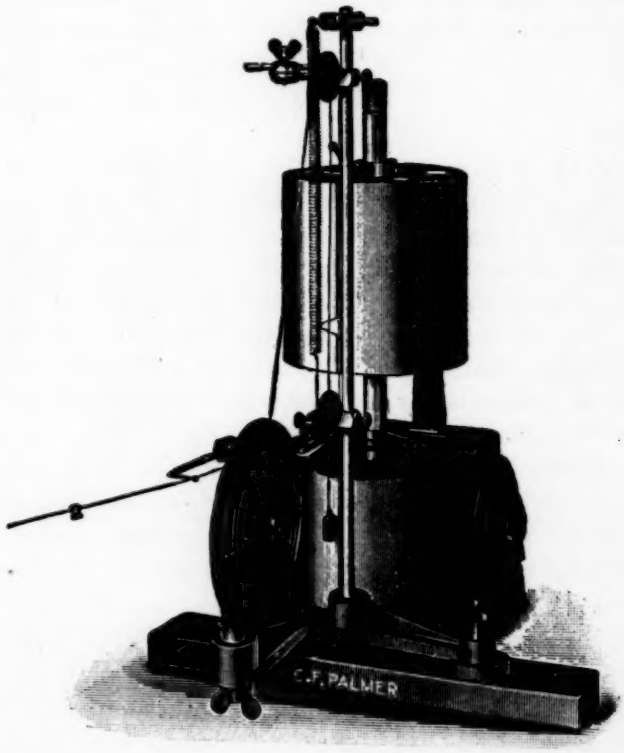


Fig. 8.—Recording kymograph.

by vertical adjustment of either of the pulleys on the upright arm. Two simultaneous records can be obtained by placing two grooved pulleys on each arm on the upright, and where the movement to be recorded is small, direct connection between subject and writing point can be made. The drum is driven by a small geared motor. The apparatus has been made by C. F. Palmer of London. The recording of the moment of stimulation in eliciting tendon or other reflexes has been described by the author in *Brain*, 37: 333, 1914.

TELANGIECTASIS OF THE SPINAL CORD*

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AND

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PHILADELPHIA

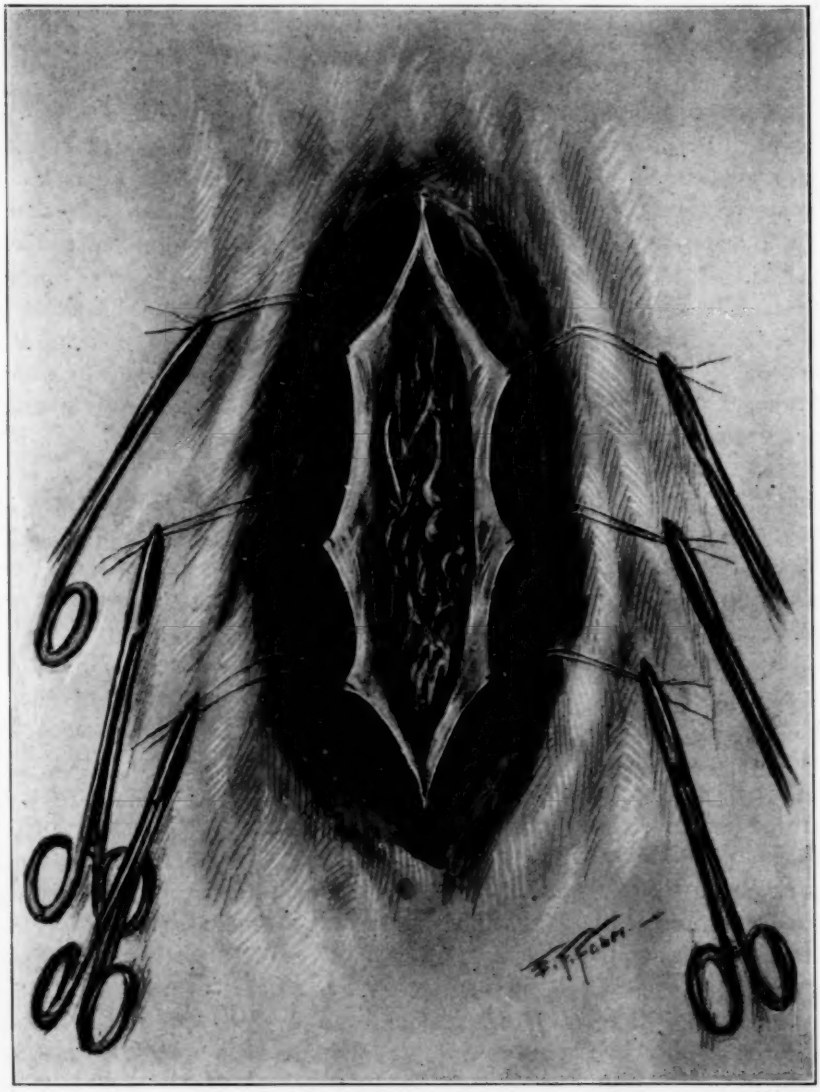
Telangiectasis of the vessels of the spinal cord or brain is not of common occurrence. It may be present many years without causing pronounced symptoms, and when in the spinal cord may be as latent as spina bifida occulta. The latter condition we have known to produce the first serious symptoms in early adult life. When no unusual demand is made on the function of the lower cord the lesion may escape detection. The activity of the sexual function may be responsible in some cases for the appearance of symptoms in early adult life. The sexual organs, as well as the bladder and rectum, have a representation in the lower sacral cord, although doubtless the sympathetic system is much concerned in the control of these organs.

The case of telangiectasis of the lower part of the spinal cord which we report is interesting, from the fact that, in 1912, symptoms suggesting myelitis developed with the rapidity common in this condition. The improvement in myelitis was unusual, although about a year was required before the patient was able to walk in an almost normal manner. It seems possible that, as a result of the myelitis, telangiectasis of the spinal vessels developed from secondary congestion, and yet for about six and a half years the woman was able to dance, swim and play tennis, and she performed these exercises strenuously with the idea of strengthening her lower limbs, but during this period her right foot had a tendency to turn inward and she walked on her right toes strongly flexed. She probably overstrained weakened nerve cells of the spinal cord, and had she been more careful she might have escaped paralysis. She married in September, 1919, and paralysis of motion and sensation in the lower limbs began again in February, 1920, and reached considerable severity. She was obliged to walk with a cane. In the hope of benefiting her surgically, operation was undertaken.

It is difficult to decide regarding ligation when one is confronted with telangiectasis of the brain or spinal cord. If too many vessels are ligated, the possibility of disturbing the nutrition of the cord must be considered. In the case reported in this paper intervention was limited to ligation of only one vessel, and even this caused temporary impairment of function. We have observed a case of telangiectasis of one cerebral hemisphere, in which a considerable number of distended

* Presented before the Philadelphia Neurological Society, Jan. 26, 1923.

vessels were ligated. Following the operation the patient developed hemiparesis, from which he only partially recovered. In another case ligation of a few vessels in telangiectasis of the cerebral hemisphere caused subcortical hemorrhage in the affected area; while in still another



Telangiectasis of Cord.

similar case ligation only sufficient to control the vessels bleeding from the operation with exposure of the brain, resulted in great improvement in jacksonian epilepsy with cessation of convulsions for several years.

The following case was referred by Dr. W. W. Richardson, of Mercer, Pa.

Clinical History.—Mrs. F. G., 25 years of age, was first seen Sept. 11, 1920. It was said that on July 3, 1912, she suddenly began to have severe pains in both lower limbs, which lasted about two hours. She managed to walk home, a distance of two or three blocks, and while doing so noticed that her lower limbs were stiff and weak; when she reached home she could scarcely walk. She was given a hypodermic injection to relieve the pain and has had no pain since that time. Tested by pinprick she was found to have great disturbance of the sensation in the lower limbs. She does not recall whether she had any elevation of temperature. She required catheterization for two weeks and was constipated. After two weeks she was able to sit in a chair and gradually learned to walk again. She became able to swim, play tennis and dance, but the right foot had a tendency to turn inward.

She remained in good health until February, 1920. She then noticed that she was getting weak and stiff in the right lower limb. The symptoms increased gradually, so that for two months before coming under observation she could hardly walk, and when first seen could only walk about one block with the use of a cane. The patellar reflexes were greatly exaggerated; the right foot showed a tendency to turn inward, but this could be overcome by passive movement; the ankle joint showed considerable flaccidity, but the muscles of the right knee were a little spastic. The Babinski reflex could not be obtained, but the Oppenheim reflex was present. Tactile and pain sensations were affected in the distribution of the sacral roots of each side. The right lower limb was spastic and the toes of the right foot were dragged along the ground in walking. The functions of the bowels and bladder were somewhat disturbed.

Operation.—An exploratory laminectomy was performed Sept. 24, 1920. The spinous processes and laminae of the twelfth thoracic and first lumbar vertebrae were removed. The dura was opened and, immediately, the lesion was exposed exactly at the level of the first lumbar segment. This was composed of a collection of convoluted varicose vessels of various dimensions, which seemed to extend well down along the right side of the cord, and to a certain extent on the left side. Whether the varicosities entirely surrounded the cord could not be determined. The largest of these vessels was ligated. In doing this the vessel was torn, but hemorrhage was finally controlled with the aid of a muscle graft. The dural wound was closed with a continuous suture and the muscles with tier sutures. Tubular drainage was used.

Course.—The patient's condition was somewhat worse for a time after the operation, but she gradually improved and when reexamined June 7, 1922, her condition was about as it had been before the operation.

The question whether it would be safe for our patient to have a child arose on account of the serious implication of the sacral region of the cord. Although the functions of the bladder and bowels are greatly disturbed by a severe lesion of the lower sacral levels of the cord, the uterine functions seem to be more independent of this region. Dr. Richard C. Norris, who has had an extensive experience covering many years in obstetrical work, informs us that he knows that a woman with advanced tabes may have a normal childbirth, although with less

labor pain; and that the same is true of a woman with severe lesion of the lower sacral region of the cord. Böwing [1] has recently reported the same result in regard to the cord lesion.

A case with a lesion similar to the one described here has been reported by Cobb,[2] who also collected the reports of seven cases in the literature. In two of the cases recurrent attacks occurred, but not with so long an interval of apparent health as in our case. It is possible that the attack in our patient which began on July 3, 1912, was really the first clinical evidence of a previously existing telangiectasis. The interval of about six and a half years between the two attacks was exceptionally long. No nevi of the skin were observed in our patient.

1. Böwing: Deutsch. Ztschr. f. Nervenheilk. **75**: Nos. 4 and 5, 1922.
2. Cobb, Stanley: Annals of Surgery **62**:641, 1915.

COMPLETE TRANSVERSE LESION OF THE SPINAL CORD WITH RETENTION OF SUPERFICIAL REFLEXES

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Bastian's law which has generally been accepted since 1882 is: Total, complete severance of the spinal cord causes absolute anesthesia to all forms of stimuli and flaccid paraplegia, with loss of all reflexes and all visceral reactions, below the site of the lesion. In this connection Collier,[1] as late as 1916, advised careful study of the plantar reflexes as offering some clue to prognosis, a progressive change from no reflex to reduced but returned reflex arguing for incomplete lesion.

There is ample clinical evidence, especially that based on observations during the late war, to cause a modification of Bastian's law, particularly in regard to the so-called superficial reflexes. Riddock[2] has shown that in complete section there is a stage of flaccidity; then a stage of reflex activity, when the bladder and rectum may act automatically (vegetative pathways), and muscle tonus may pick up; and then a gradual failure of all reflex activities.

Head[3] states "when the spinal cord has been completely divided, without widespread destruction or septic infection, the lower end may, under favorable conditions, regain its tonic influence and reflex excitability. As shock disappears its various functions gradually return; but they now assume a form in which the reflexes have lost, to a great extent, their local signature, and the reaction is massive rather than specific."

Guillian and Barré[4] have made an exhaustive study of the reflexes in fifteen cases of complete lesion of the spinal cord due to war injuries, the lesions being located between the last cervical and last dorsal segments, and offer the following conclusions: (1) Complete lesion is not necessarily accompanied always at first by muscular hypotonia. (2) The tendon reflexes are usually abolished and remain so until death. (3) The plantar cutaneous reflex is almost always present, but

1. Collier, James: Lancet **1**:711 (April 1) 1916.

2. Riddock, George: "The Reflex Reactions of the Completely Divided Spinal Cord in Man, Compared with Those Associated with Less Severe Lesions," Brain **40**:264, 1917.

3. Head, Henry: Studies in Neurology, Oxford University Press, 1920.

4. Guillian, George, and Barré, J. A.: Anatomical Clinical Study of Fifteen Cases of Complete Lesion of the Spinal Cord, Ann. d. Méd. de Paris, **4**:178, 1917.

is sluggish and tardy; it differs somewhat from the normal type. (4) Reflexes of defense, sought by classic methods, are rare, very weak, and are only occasionally produced by stimulation of the dorsum of the foot; stimulation on the sole of the foot, on the contrary, gives in the same paraplegias diffuse reactions which have the same characteristics as reflexes of defense, and are observed in more than half the cases. (5) Cremasteric reflexes are often preserved. (6) Abdominal cutaneous reflexes are rarely observed. (7) Anesthesia is total in all senses, namely: touch, pain, temperature, etc. At the upper limit of anesthesia there is dissociation of sensations. (8) Retention of urine is the rule. (9) Incontinence rather than retention of feces is more often observed. (10) The extremities are usually warmer than normal, the temperature being higher toward the feet.

REPORT OF CASES

I wish to report briefly three cases which were observed during my service as neurological surgeon to Mobile Hospital No. 1, during the St. Mihiel offensive. In each case, one or more of the superficial reflexes were retained below the level of the lesion, although at necropsy the cords disclosed complete transverse lesions.

CASE 1.—W. S. was admitted Sept. 16, 1918, 31 hours after being wounded. He was in a state of extreme shock. The pulse was very rapid and hardly perceptible. There was a perforating wound of the neck at the level of the fourth or fifth cervical segment.

Neurologic Findings: Loss of sensation existed below the level of the second rib on both sides, and there was loss of sensation in both arms. Motor paralysis was complete in both forearms and hands, and in both legs. Breathing was of purely abdominal type. No contraction of the intercostal muscles occurred during respiration. There was loss of all deep and superficial reflexes below the level of the lesion with the exception of the cremasteric reflexes which were present both on the right and the left. There was no paralysis of the cervical sympathetics.

Death occurred Sept. 17, 1918.

Necropsy Findings: Lesion of the spinal cord with complete destruction of the fourth and fifth cervical segments. (Specimen A in accompanying illustration.)

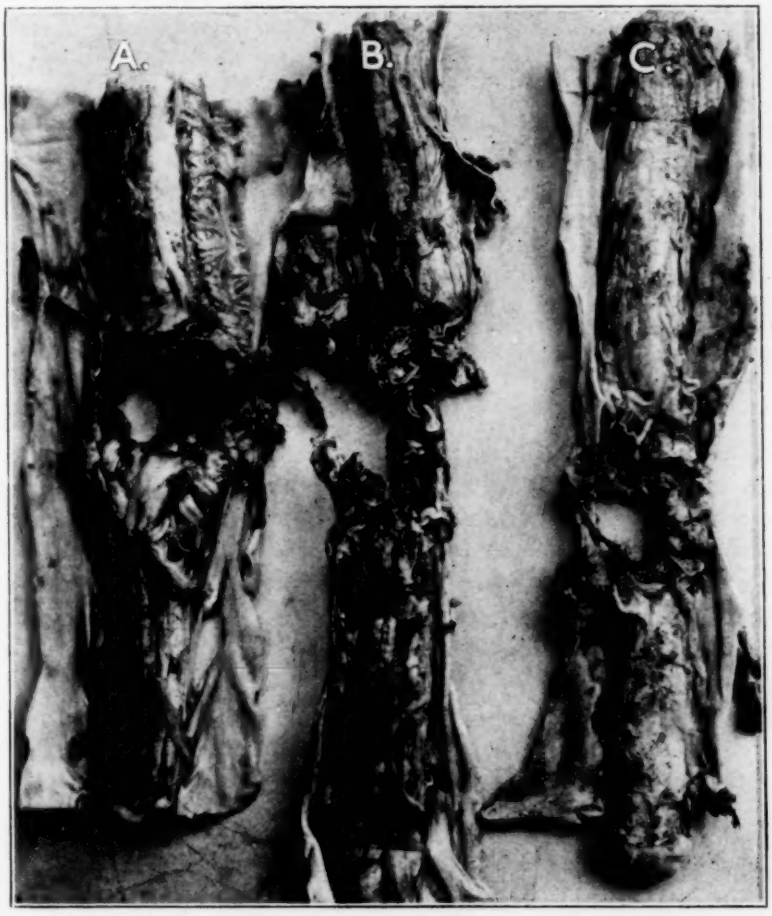
CASE 2.—G. Y., was admitted Sept. 19, 1918, 28 hours after being wounded, in a condition of severe shock. A penetrating wound 2 cm. in diameter was present just to the left of the fifth thoracic vertebra. A watery discharge was observed from the wound which was either cerebrospinal or pleuritic fluid. Roentgen-ray examination disclosed a shell fragment, 4 by 12 mm., which moved with respiration, in the left side of the mediastinum and a pneumothorax on the right side.

Neurologic Findings: There was complete loss of all sensibility below the sixth rib on both sides. All deep reflexes were absent below the lesion. Epigastric, abdominal, cremasteric and plantar reflexes (flexor type) were active both on the right and left sides. There was loss of bladder and rectal

control. Priapism was present. There was a zone of hyperesthesia above the upper limit of anesthesia, 6 cm. wide on the left and 10 cm. wide on the right.

Operation: Débridement of the wound and laminectomy were performed 35 hours after the injury. The spinal cord at the level of the sixth thoracic segment was found completely divided. Death occurred Sept. 21, 1918.

Necropsy: The spinal cord was completely divided at the level of the sixth and seventh thoracic segments (Specimen B). There was right pneumohemo-



A. Spinal cord from Case 1. B. Spinal cord from Case 2. C. Spinal cord from Case 3.

thorax; and a shell fragment, 4 by 12 mm., was lodged in the wall of the right bronchus just to the right of the bifurcation.

CASE 3.—M. R., was admitted Sept. 14, 1918, approximately 24 hours after being wounded, in a condition of severe shock. There was a penetrating wound in the mid-back region at the level of the third thoracic vertebra. Roentgen-ray examination disclosed a small shell fragment 4 cm. deep in the third dorsal region.

Neurologic Findings: Complete motor and sensory paralysis was present below the level of the third dorsal segment; there was absence of all deep reflexes in the legs, and absence of the plantar reflexes. Abdominal and cremasteric reflexes were present on both sides.

Operation: Débridement of the wound and laminectomy were performed. The spinal cord was found apparently completely divided at the second dorsal segment. The shell fragment was removed from the body of the second dorsal vertebra anterior to the cord. Death occurred Sept. 16, 1918.

Necropsy: The spinal cord was found practically divided at the level of the third dorsal segment. A small portion of the cord seemed intact on the left side. (Specimen C.)

The specimens from the three cases were sent to Dr. W. G. Spiller, Philadelphia, for examination. The accompanying photographs were kindly furnished by him. Following is Dr. Spiller's report: "The three cords are so completely destroyed to the naked eye at the level of the lesion that it is useless to make microscopic sections to prove this. This destruction is well shown in the photographs. The only doubt could be as regards cord C, and that doubt could exist only from examining the photograph. The specimen shows such complete destruction that it is useless to make sections for the microscope to prove complete division of the cord. The tissue present at the level of the lesion is disintegrated. Certainly no reasonable person after looking at the photographs could hold that continuous nerve paths existed at the level of the lesion in cords A and B."

SUMMARY

Three cases are reported, with necropsies, in which there was complete severance of the cord at the levels of the fourth and fifth cervical segments, sixth and seventh thoracic segments, and third thoracic segment respectively. In spite of this there was retention of the following superficial reflexes in the parts below the level of the lesion: cremasteric reflexes on both sides; epigastric, abdominal, cremasteric and plantar reflexes (flexor type) on both sides; abdominal and cremasteric reflexes on both sides.

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THE DUAL NATURE OF THE EFFERENT NERVOUS SYSTEM

A FURTHER STUDY OF THE STATIC AND KINETIC SYSTEMS, THEIR FUNCTION AND SYMPTOMATOLOGY *

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IV. COMMENTS AND CONCLUSIONS.

In my presidential address to the American Neurological Association, in June 1920,[1] I considered the efferent nervous system from a new point of view. In this conception, two distinct systems of motility were

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* Amplification of a paper presented to the American Society for Clinical Investigation, May 1, 1922; preliminary reports to the American Neurological Association, June 15, 1921, and the New York Neurological Society, June, 1922.

1. Hunt, Ramsay: The Static and Kinetic System of Motility, Arch. of Neurol. & Psychiat. 4:353 (Oct.) 1920.

recognized throughout the whole of the efferent nervous system, subserving respectively the functions of motion and posture. While the generalization which I then formulated was applied to the whole efferent mechanism, my argument was concerned chiefly with the cerebrospinal nervous system and voluntary muscles. In the present study I propose to elaborate this subject still further, giving special consideration to the static and kinetic systems in the vegetative nervous mechanism and involuntary muscle. As an introduction to my subject, and before proceeding to a discussion of the involuntary nervous system, I will review briefly my views regarding the static and kinetic components of the cerebrospinal system and their relation to the symptomatology of motor disorders.

I. THE CEREBROSPINAL NERVOUS SYSTEM

1. THE STATIC AND KINETIC COMPONENTS

The function of motility may be resolved into two components, each represented throughout the entire efferent nervous system by separate neural mechanisms, which are physiologically and anatomically distinct. One of these components is the movement proper, which is subserved by the *kinetic* system. The other component is that more passive form of contractility, underlying postural tone, and is subserved by the *static* system. The term static is used here to designate that peculiar property of muscle by which it becomes fixed in posture.

The field of motility in animal life is generally divided by physiologists into three large groups: reflex, automatic-associated, and isolated-synergic types of movement. These various types of movement, although showing very distinct differences, merge by imperceptible gradation into one another.

In the lowest forms of life, movement and posture are of a purely reflex character and depend on a primitive reflex arc, which is the functional unit of the nervous system (*archeokinetic* and *archeostatic* systems). In higher forms these reflex units are combined and more complicated reflex activities result, and these in turn are gradually brought under the control of still higher integrating and correlating centers with corresponding advances in the variety and forms of movement and of posture; it may be stated as a general principle that there is no form of movement from the simple reflex of the vegetative nervous system to the highest type of cortical expression which functions without a corresponding static mechanism.

Generally speaking, both from the anatomic and physiologic standpoint, the central nervous system may be said to represent three great structural and functional divisions. One is the segmental nervous system which contains the great reflex systems of the neuraxis; and is the archaic representation in man of the nervous reactions of the lowest

forms of life. The other is the paleo-encephalon represented by precursors of the corpus striatum and optic thalamus which constitute the higher coordinating sensory and motor mechanisms of the lower forms of life. The neo-encephalon represents the latest stage in the evolution of the nervous system and has its greatest development in man.

These three functional divisions of the nervous system are related to the three great types of movement and posture already mentioned, and are represented by corresponding kinetic and static mechanisms.

There are, for example, *archeokinetic* and *archeostatic* systems subserving the function of reflex movements and postures of the segmental nervous system. There are also *paleokinetic* and *paleostatic* systems subserving the function of automatic-associated movements and postures; and *neokinetic* and *neostatic* systems subserving the function of isolated-synergic types of movement and posture.

In this rather schematic division of motility and its mechanisms it should be understood that the development from one type of movement to another, and from one structural division to another, is a gradual one. It represents a transition of structure and function which may be traced in the phylogenetic history of animal life and the imprint of which remains stamped on the central nervous system of man.

2. ANATOMIC CONSIDERATIONS

1. *The Static System.*—The essential integrating and correlating mechanism for the control of the static or posture function of motility, I believe to be the cerebellum.[2] Afferent impulses from the periphery and efferent impulses from the cerebral cortex pass to this organ before their final distribution by way of the cerebellospinal systems to the myostatic or posturing mechanism of skeletal muscles.

Edinger pointed out some years ago that the cerebellum presents phylogenetic evidences of two great divisions, which correspond to similar divisions of the cerebrum. The paleocerebellum receives impulses which pass from the spinal cord to the vermis, and constitute the vermian system. The neocerebellum receives impulses from the cerebral cortex, which pass by way of the pons varolii to the cerebellar hemispheres, forming the hemispheric system.

The paleostatic system, which controls the postural functions of automatic and associated type, takes its origin in the older nuclei of the vermis cerebelli (nucleus fastigius, globosus, and emboliformis).

The cerebellar hemispheres, on the other hand, regulate the higher postural functions of motility through the medium of a neostatic system, which takes its origin in the cells of the dentate nucleus. The

2. Hunt, Ramsay: The Static or Posture System and Its Relation to Postural Hypertonic States of the Skeletal Muscles, Neurol. Bull. **3**:207, 1921.

neostatic system is controlled from the cerebral cortex by special tracts which pass to the nuclei of the pons varolii, and thence to the opposite hemisphere of the cerebellum.

It is probable, on phylogenetic grounds, that the parietotemporo-occipital tract which terminates in the dorsal pontine nuclei is older than the frontopontine system which descends from the frontal lobe to the ventral nuclei of the pons. This latter system I regard as the homologue in the static mechanism of the corticospinal or neokinetic system. The frontopontocerebellar or neostatic system descends in the anterior limb of the internal capsule and the mesial portion of the cerebral peduncle in close relationship with the pyramidal tract, and it is interesting to note that both of these structures, the one subserving the neostatic and the other the neokinetic functions of motility, receive their myelin sheaths subsequent to birth.

The other corticocerebellar systems (parietooccipitotemporal systems) pass in the lateral portion of the crus cerebri to the dorsal pontile nuclei, and thence to the opposite cerebellar hemisphere. I believe that they represent corticocerebellar connections subserving higher types of posture in connection with the reflex activities of the various sensory areas of the cerebral cortex. They are the homologues in the static sphere of the corticothalamic connections with the striospinal or paleokinetic system.

The chief efferent systems of the cerebellum (paleostatic and neostatic) pass by way of the superior peduncles to special cellular divisions of the nucleus ruber. The paleocerebellar system terminates in the nucleus magnocellulatus and the neocerebellar system in the nucleus parvicellulatus. From here they descend in the rubrospinal and other systems of the spinal cord, then passing to the sarcoplasm of the skeletal muscles as peripheral static systems. The distribution of the static system in peripheral nerves and its mode of termination in the muscle fiber is still one of the mooted questions in histology and physiology. There is, however, considerable evidence in favor of the dual innervation of the striated fiber, but anatomists differ widely as to the manner in which these peripheral static or posture systems reach the muscles. When, however, one considers the well developed central mechanism for the dual functions of the efferent system, there is little doubt as to the existence of a corresponding difference of structure and function in peripheral nerves. Ransom [3] has already demonstrated, by a special method of staining, the existence of large numbers of non-medullated nerve fibers in peripheral nerves, and perhaps here is concealed the solution of this intricate question.

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3. Ransom: Unmyelinated Nerve Fibers as Conductors of Protopathic Sensation, Brain **38**:381, 1915.

It is interesting to observe that all the posture systems, neostatic as well as paleostatic, pass to the cerebellum for final integration and coordination. This is in accord with the nature of the posturing mechanism and its secondary and unconscious rôle in motility. For while the higher forms of movement are initiated as conscious and voluntary processes, the corresponding postures are secondary, and follow automatically in the path of movement. For this reason, the neostatic system does not pass directly to the gray columns of the spinal cord, as do the pyramidal tracts (neokinetic system) but is directed first to the cerebellum, which is the great central correlating mechanism for posture function. This aspect of the static system I have already considered in a study of the relation of the cerebellum to the posture synergies of movement.[4]

2. *The Kinetic System.*—The kinetic component of motility is concerned with the transmission of impulses underlying movement. As has already been mentioned, the primitive reflex mechanisms subserving movement are represented in the archeokinetic systems of the segmental nervous system. The higher motor centers for the control of kinetic function are represented in the corpus striatum and the rolandic areas of the cerebral cortex.

In a study of paralysis agitans[5] I expressed myself as favoring the conception of two great motor systems from brain to muscle, one subserving paleokinetic and the other neokinetic functions. The corpus striatum, according to this view, is the paleokinetic center for the control of "automatic and associated movements," and the rolandic area is the neokinetic center for dissociated synergic movements of cortical origin. Their static functions, as has already been indicated, I believe are subserved by other pathways.

It is interesting to note, however, that the thalamic portion of the paleo-encephalon is closely associated with the cerebral cortex by both afferent and efferent tracts. It is, I believe, through the medium of these tracts, and the optic thalamus that paleokinetic motility is controlled from the cortical sphere (corticostriospinal system). This higher control of the striospinal system makes possible a finer differentiation of paleokinetic motility, and would explain the occurrence of purely involuntary movements of cortical origin.

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4. Hunt, Ramsay: Dyssynergia Cerebellaris Myoclonica (Primary Atrophy of the Dentate System). A Contribution to the Pathology and Symptomatology of the Cerebellum, Brain **44**:490, 1921.

5. Hunt, Ramsay: Progressive Atrophy of the Globus Pallidus: A System Disease of the Paralysis Agitans Type, Characterized by Atrophy of the Motor Cells of the Globus Pallidus Mechanism. A Contribution to the Functions of the Corpus Striatum, Brain **40**:58, 1917.

The existence of these intermediate kinetic and static systems connecting the various sensory areas of the cortex with the thalamostriate mechanism on the one hand (corticopaleokinetic system), and the pontocerebellar mechanism on the other, are confirmatory of my view of the parallelism of structure and function which underlies the development of motion and posture.

The neokinetic system is in striking contrast to the paleokinetic system. It is a direct pathway from the cerebral cortex to the spinal cord for the control of isolated synergic movement. It takes its origin in the motor cells of the rolandic area and passes by way of the pyramidal system directly to the motor cells of the brain stem and spinal cord; and from there to special disk representations in muscles, subserving the dissociated movements of cortical origin.

These two great motor systems, the neokinetic and paleokinetic, according to this theory, have both a functional representation in nerves and skeletal muscles. They are in a sense complementary and their independence is only unmasked by the dissociations of disease. The neokinetic and paleokinetic system I regard as the functional counterpart in the motor sphere of Head's epicritic and protopathic systems in the realm of sensation.

A critical analysis of the symptomatology of pyramidal and extrapyramidal disease (spastic paralysis and paralysis agitans) are strongly corroborative of this hypothesis.[6]

Prespinal or Supraspinal Centers of Motion and of Posture.—In the crura, pons and medulla oblongata are found a number of important ganglionic structures of rather obscure function. Some years ago, the motor centers of this region were grouped together by Edinger under the general heading of nucleus motorius tegmenti (nucleus ruber, Deiter's nucleus).

These motor centers form the connecting link between the cerebellum, on the one hand, and the corpus striatum, on the other, by means of which these organs exercise their function on the skeletal musculature.

I have already presented the evidence showing that the efferent pallidal system of the corpus striatum represents an internuncial common path for the transmission of motor impulses from the striatum to certain ganglionic structures of the midbrain and subthalamic region, in the interest of automatic-associated movements.[7]

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6. Hunt, Ramsay: The Existence of Two Distinct Physiological Systems for the Transmission of Motor Impulses in Peripheral Nerves, Brain **41**:302, 1918.

7. Hunt, Ramsay: The Efferent Pallidal System of the Corpus Striatum. A Consideration of Its Functions and Symptomatology, J. Nerv. & Ment. Dis. **46**:44, 1917.

In the same manner the cerebellum by means of its efferent dentate system sends fibers subserving a postural function to the nucleus ruber and other nuclear structures of the midbrain and medulla.

The essential point in my conception of the functional relationship of these systems is as follows: The pallidal system of the striatum is kinetic and passes to nuclear structures in which are represented motion formulas, while the dentate system of the cerebellum has a static function and passes to nuclear structures in which are represented posture formulas. In other words the motor centers of the prespinal mechanism which play so important a role in motility have both a kinetic and static representation, which in functional terms may be expressed by motion and posture formulas.

3. *Peripheral Myostatic and Myokinetic Systems.*—The striated muscle fiber is composed of two distinct substances, one subserving a contractile, the other a postural function. The sarcostyle is the contractile portion of the muscle fiber and represents the myokinetic mechanism; the sarcoplasm is a more homogeneous substance and represents the postural or myostatic mechanism. In a general way it is estimated that the sarcostyle constitutes from one sixth to one half of the total muscle mass, the remaining portion of which is sarcoplasm. Each striated fiber contains a motor nerve ending, which is the terminal of a medullated nerve fiber. For many years this was thought to be the sole innervation of the muscle fiber, until Perroncito [8] and Boeke [9] demonstrated the existence of another smaller nerve ending in the striated muscle fiber, the terminal of a non-medullated nerve.

Boeke expressed the opinion that the motor end plate of the medullated nerve fiber furnished the stimulus for the disk system while the accessory non-medullated fiber was in control of the sarcoplasmic portion of the contractile mechanism, a conception which Mosso [10] had previously expressed in his interpretation of the tonus and twitch components of muscle function.

It is interesting to mention that physiologists have also detected certain differences in the metabolism of muscles which are probably related to these two forms of activity. Pekelharing [11] demonstrated the

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8. Perroncito: Études ultérieures sur le terminaison des nerfs dans les muscles à fibres striées, Arch. Ital. d. Biol. **38**:393, 1902.

9. Boeke: Die motorische Endplatte bei den höheren Vertebraten, Anat. Anz. **25**:193 and 481, 1909.

10. Mosso and Pellacani: Sur les functions de la vessie, Arch. Ital. d. Biol. **1**:97 and 291, 1882.

11. Pekelharing and Van Hoogenhuyze: Die Bildung des Kreatins im Muskel beim Tonus und bei der Starre, Ztschr. f. physiol. Chem. **64**:262, 1910; Pekelharing: Die Kreatininausscheidung beim Menschen unter dem Einfluss von Muskeltonus, Ztschr. f. physiol. Chem. **75**:207, 1911.

existence of two chemical processes in muscle metabolism. One was related to the disk system, and involved the utilization of non-nitrogenous substances. The other was concerned with the utilization of albuminates and was probably dependent on the chemical activity of sarcoplasm. Other experiments have shown that the expenditure of energy of the active muscle contraction (tetanus) is far greater than is that of posturing muscle (tonus) which explains the relative unfatigability of the posturing or sarcoplasmic function of muscle. It is certainly significant that muscle fibers which consist of two distinct types of contractile substance should also present two distinct forms of chemical activity, under different functional conditions.

Comparative anatomy also shows clearly that the muscular system, like all other structures of the body, is in a state of evolution, adapting itself to the changing conditions of the organism. Development is from the slow movement of the unstriped to the quick movement of the striated muscle fiber. And in man all gradations may be observed from the lowest type of unstriped muscle to the highest type of striated muscle fiber. This subject will be discussed more in detail in a subsequent chapter dealing with unstriped muscle and the vegetative nervous system.

A clear conception of the phylogeny of contractile tissue is important to the theory of a dual system of motility, as the skeletal muscle mass in man is not to be regarded as a mere aggregation of striped or unstriped muscle fibers of standard type; but rather as systems of contractile or effector end organs representing various stages of phylogenetic development, and thus corresponding in some measure to the phylogeny of nerve tissue.

I believe that the higher functions of motility, in both voluntary and involuntary muscle, are carried out by more highly developed muscle fibers than are lower forms of movement. The neokinetic function is associated with a higher type of muscle fiber and more finely differentiated disk mechanism than are those subserving archeokinetic and paleokinetic functions. The efferent nervous system and muscle tissue are so closely associated that evolutionary progress in one proceeds with corresponding changes in the other, to meet the demands of more elaborate function activity.

3. THE RELATION OF THE STATIC AND KINETIC COMPONENTS OF THE CEREBROSPINAL NERVOUS SYSTEM TO SYMPTOMATOLOGY

I will now pass to a consideration of the relation of the static and kinetic systems to the various disorders of motility. It may be stated, as a general principle of symptomatology, that a lesion of the kinetic system causes a disorder of movement, and injury of the static system

a disorder of posture. Under certain conditions there are apparent exceptions to this rule. For example, an injury to the cerebellum may cause a disorder of movement (cerebellar dyssynergia — intention tremor). These motor manifestations, however, are merely secondary to the loss of postural control and represent compensatory manifestations in the kinetic sphere. Therefore, loss of the static mechanism with consequent disturbances of postural control, may, by reason of secondary kinetic manifestations, simulate very closely a primary disorder of the motion system.

In nearly all disorders of motility both systems participate, although it is often possible to indicate one or the other as the primary or essential factor involved.

At the reflex level of the nervous system, the tendon reflex is a typical example of kinetic function as is reflex posture of the static function. Exaggeration of the tendon reflexes and muscle clonus, (ankle clonus, patellar clonus and wrist clonus) are referable to disorders of the myokinetic mechanism. On the other hand exaggerations of postural tone and reflex postural activity are referable to the myostatic mechanism. Foix [12] has recently considered the question of postural reflexes and plastic contractures in relation to certain organic nervous diseases.

It has been shown by Langelaan [13] that tonus consists of two components, a contractile tonus referable to the sarcostyles, and a plastic tonus of sarcoplasmic origin.

By an analysis of the extension curves of tonic muscle, Langelaan reached the conclusion that the tonic muscle is continually in a state of slight contraction, combined with a state of increased plasticity. The source of the stimuli which maintain this peculiar state of the muscle are the movements of the body which excite continuously the proprioceptors of the whole apparatus of locomotion. These physiologic facts he brings into association with the morphologic discoveries of Boeke,[9] and the conclusion is reached that plasticity is the chief property of the sarcoplasmatic part of the muscle, while the maintenance of a slight state of contraction is due to the striped apparatus. The plastic component of tonus, according to Boeke, is controlled by the sympathetic system, and the contractile component is under the control of the motor cell of the anterior horn. Of these two components of tonus, the contractile is the more variable. The plasticity or autonomic component is withdrawn from voluntary control and is the more stable factor in muscle tone.

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12. Foix, M. C.: Réflexes tonique de posture, Rev. neurol. **28**:1130, 1922.
13. Langelaan, J. W.: On Muscle Tonus, Brain **38**:235, 1915.

In accordance, therefore, with the conception of the duality of function of the efferent system, contractile tonus is related to the kinetic system, and plastic tonus to the static system. The former may be termed *kinetotonus* and the latter *statotonus*, thus indicating their dependence on these two fundamental systems for the regulation of their contractile function.

These two divisions of the contractile mechanism may also be recognized on direct percussion of the muscle. The kinetic component may be distinguished by the quick contractions of myotatic irritability and the static by the more persistent local reactions of the idiomuscular response (myedema). Direct percussion of muscle in myotonia shows very clearly the prolongation of the local reaction due to the persistence of the contraction of the sarcoplasm.

Spasmodic disorders of motility, such as chorea, paramyoclonus multiplex, myokymia, and fibrillary twitchings are referable to the kinetic mechanism. These spasmodic manifestations in the kinetic sphere occur as a result of irritation, or as release phenomena in subordinate motor systems due to loss of higher inhibitory control.

The clonus of spastic paralysis (ankle, patellar and wrist clonus) and the tremor of paralysis agitans are both of kinetic origin and referable to the kinetic system. In a study of organic tremor, I have already considered the relation of the tremor of paralysis agitans (striatal tremor) to the kinetic mechanism, and that of cerebellar origin (cerebellar tremor) to the static mechanism.[14] The striatal tremor occurs spontaneously, is rhythmical and is associated with lesions of the corpus striatum which release the subordinate motor centers of the extrapyramidal system from control.

The cerebellar tremor, on the other hand, is not spontaneous but occurs only on movement and is of the so-called intention type. It is due to a loss of the postural function of the cerebellum and is exaggerated by the compensatory activities of the kinetic mechanism. A combined form of striocerebellar tremor also occurs in multiple sclerosis, pseudosclerosis and other organic affections from combined involvement of these two systems in the cerebellum and the corpus striatum or their point of junction in the midbrain.

In both the clonus of spasticity and the tremor of paralysis agitans there is loss of inhibitory control of higher neural mechanisms. In spastic clonus the cerebral inhibition of the spinal centers is abolished by a lesion of the pyramidal tract (corticospinal systems). In the tremor of paralysis agitans there is a loss of striatal inhibition of the extrapyramidal tracts by lesions of the corpus striatum. In both of

14. Hunt, Ramsay: The Strio-Cerebellar Tremor (Trans. Am. Neurol. Assoc. 1922) Arch. Neurol. & Psychiat. **8**:664 (Dec.) 1922.

these forms of central palsy there are secondary exaggerations of the plastic component of muscle tone.

Postural Hypertonicity of the Skeletal Muscles.—Postural hypertonicity of the skeletal muscles bears a definite relation to the static system and the sarcoplasmic function of muscle.[2] Many different types of postural hypertonus following paralysis may be recognized, which depend on the nature and localization of the lesion within the central nervous system. The more important of these are the spasticity of pyramidal tract disease; the musclar rigidity of paralysis agitans; the decerebrate rigidity of midbrain section, and the postural hypertonic effects of transverse lesions of the pons, medulla and spinal cord.

Cortical or Pyramidal Types of Postural Hypertonus (Spasticity): One of the most characteristic symptoms of organic disease of the central nervous system is spastic hypertonicity of the skeletal muscles. The affected muscles, while weak or paralyzed, are in a state of postural fixation which is dependent on the release and overactivity of subordinate kinetic and static centers.

Any injury of the neokinetic or corticospinal system is followed by paralysis of the isolated-synergic movements of cortical origin and release of the lower motor mechanism of the spinal cord. There is a kinetic discharge which is accompanied by postural fixation. Paralysis of the corticospinal or neokinetic system releases the subordinate neokinetic spinal mechanism which is especially concerned with this type of motion.

It is for this reason that the chief postural deformities of the spastic state are so sharply localized in the distal portion of the extremities, and more especially the hands and fingers. For the hand is the chief instrument of the higher neokinetic activities of man, and therefore shows the greatest degree of palsy in hemiplegia of the pyramidal type.

The tendency to clonus, the exaggerated tendon and periosteal reflexes in spastic states, I would also explain by the heightened reflex activities of the spinal cord. The corticospinal system passes directly to the reflex mechanisms of the spinal cord, in the interest of the dissociation of movement, and any diminution or loss of this inhibitory control releases the neospinal mechanism, which explains the increased reflex activity of tendon and muscle response.

A further elaboration of this question will be found in an earlier study of neokinetic and paleokinetic representations in peripheral nerves.[6]

Striatal or Pallidal Types of Posture Hypertonus (Paralysis Agitans Rigidity): The rigidity of paralysis agitans is a postural hypertonicity resulting from paralysis of the pallidal system of the striospinal pathway. In previous studies of paralysis agitans this point of view has

been presented, together with evidences showing that the efferent motor system of the corpus striatum (pallidal system) represents an internuncial common pathway for the passage of motor impulses for automatic-associated types of movement (paleokinetic system).[13]

The characteristic attitude and postural deformities of this affection present many points of difference from those associated with the spastic state. Furthermore, the hypertonicity is more of the plastic waxy type, and lacks certain elements which characterize the spastic state. This I would explain by the peculiar location of the lesion in the pallidal system of the corpus striatum. Paralysis of this system releases from inhibition the various kinetic centers of the midbrain and hypothalamic region, among which may be mentioned such structures as the red nucleus, and the substantia nigra. When these subordinate centers of the paleokinetic system are released, their various motion-complexes are released and become fixed in postures.

It will be recalled, however, that the hypertonicity of paralysis agitans is not accompanied by clonus and other evidences of exaggeration of the reflex activities. The reason for this is obvious. The lesion of paralysis agitans releases only the kinetic centers of the midbrain and hypothalamic region, and the reflex kinetic centers of the spinal cord are still controlled at this higher level. Whereas, in lesions of the corticospinal pathway (pyramidal tracts), all central inhibition of the lower spinal mechanism is abolished, with the development of the characteristic reflex activities of the spastic state.

Another point of interest is the tremor of paralysis agitans, which is so characteristic of the pallidal type of palsy. This tremor is also the expression of subordinate kinetic centers of the hypothalamic and midbrain region which are under the control of the corpus striatum, and which when released express themselves in terms of tremor. It is not unlikely that the striatal connections with the red nucleus are closely related to this form of tremor; but here we are still in a very conjectural field. It would appear probable, however, that the red nucleus in addition to its static component, which stands in relation to the cerebellum, has also a kinetic component which is under the control of the corpus striatum. This phase of the subject I have considered more in detail in my contribution to the combined form of striocerebellar tremor.[14]

Pallidopyramidal Types of Postural Hypertonus (Spastic Rigidity): The proximity of the corpus striatum (pallidal system) to the internal capsule (pyramidal system) is such that simultaneous involvement of

15. Hunt, Ramsay: Primary Atrophy of the Pallidal System: A Contribution to the Symptomatology and Pathology of Paralysis Agitans, Arch. Int. Med. **22**:647, 1918.

both structures is not uncommon. Indeed, in vascular lesions of the basal ganglions, it is probable that pure forms of either pyramidal or pallidal palsy are quite rare and that a combination of the two types is more common. Involvement of both systems, the pallidal and pyramidal, produces a spastic-rigid state which combines in varying degrees the characteristics of both types of postural rigidity.

In the combined or pallidopyramidal types of palsy there is not only loss of isolated-synergic movements of cortical origin, but also of the automatic-associated movement, referable to the striatal mechanism. With this combined form there is also a corresponding fusion of their postural elements which to my mind explains the variety of postural types in the ordinary vascular hemiplegia (pallidopyramidal type of hemiplegia).

The special feature of this fusion of hypertonicity is the combination of the waxy rigidity of paralysis agitans and the characteristic symptoms of the spastic state. Indeed, all degrees of variations may be observed in the combined types of hemiplegia, depending on the relative loss of the two great types of movement, automatic-associated and isolated-synergic, and the corresponding postural changes.

Midbrain or Paleocerebellar Type of Postural Hypertonus (Decerebrate Rigidity): Another type of hypertonicity which has awakened considerable interest and investigation is the decerebrate rigidity of Sherrington. This is a peculiar state of muscular rigidity which is produced by a section through the midbrain at the level of the anterior colliculi. A mesencephalic transection at this level severs the pyramidal tracts (neokinetic system) and the extrapyramidal tracts (paleokinetic system), as well as the corticopontocerebellar tracts (cortical static systems). The various subordinate centers of the pons, medulla and cord, both kinetic and static, are thus released and the skeletal muscles assume the postural rigidity of standing which is characteristic of this level.

As the older static system of the cerebellum for the control of posture is still functioning, retaining its afferent connections with the spinal cord and the efferent with the red nucleus, the automatic posture of standing, characteristic of decerebrate rigidity is maintained.

Spinal Types of Postural Hypertonus: Certain spinal types may also be recognized. There is, for example, the paraplegia in flexion and the paraplegia in extension. A transverse lesion of the cord would sever all of the long projection systems, both static and kinetic, and thus release its various intrinsic reflex mechanisms. In response to this loss of the higher inhibitory control, the characteristic reflex of flexion would develop, which then becomes fixed in postural hypertonus, the extreme of which is "paraplegia in flexion."

An extensor type of spinal paralysis is also encountered (paraplegia in extension). This form is characteristic of pyramidal tract disease and is merely the spinal expression of the pyramidal type of hypertonus, as observed in purely spastic states.

Myotonia: The disorder of motility which is termed myotonia, whether cerebral, cerebellar, spinal or peripheral in its origin is, I believe, referable to the static mechanism. A disorder of the myostatic mechanism is responsible for the peculiar character of myotonia, viz; the persistence of the contraction, and the inability to relax muscles after the contraction. There is a more or less persistent state of postural fixation which is not subject to the niceties of neural control and thus interferes with the course of movement itself.

Gregor and Schilder,[16] in their study of myotonia were able to demonstrate the presence of two distinct contraction waves in the electromyogram; the one quick and referable to the disk system, the other slow, more sustained and probably of sarcoplasmic origin.

The relationship of the frontopontocerebellar system to various myotonic phenomena has already been emphasized by Kleist[17] and by Kinnier Wilson.[18] Kleist mentions the occurrence of myotonia in myelitis and syringomyelia, with the typical mechanical and electrical myotonic response of the peripheral localization. There are other central types of myotonia, however, in which the mechanical and electrical responses are wanting and in which the myotonia is not diminished by repetition of the movement. Myotonia of this type he has observed in Friedreich's ataxia and Marie's cerebellar ataxia, also in certain forms of myotonia atrophica. He refers also to adiadokokinesis and catalepsy as a form of myotonic disorder, as well as the tonic perseveration and considers their possible relation to the frontopontine system.

Kinnier Wilson and Walshe[18] have also made the important observation that tumors of the frontal lobe anterior to the motor area may produce myotonia, a peculiar feature of which is the limitation of the disturbance to voluntary movements. Bumke[19] has also described a family disorder characterized by the occurrence of myotonia with both voluntary and automatic types of movement. In these central forms

16. Gregor and Schilder: Zur Theorie Der Myotonia, Neurol. Centrlbl. **32**:85, 1913.

17. Kleist: Ueber nachdauernde Muskelkontraction, J. f. Psychiat. u. Neurol. **10**:116, 1907-1908.

18. Wilson, S. A. K., and Walshe, F. M. R.: The Phenomena of Tonic Innervation and Its Relation to Motor Apraxia, Brain **37**:199, 1914.

19. Bumke: Eine familiäre Form von Intentionskrampf, Ztschr. f. Neurol. u. Psychiat. **4**:644, 1910.

of myotonia the usual electrical and mechanical evidences of myotonia congenita are absent, which proves the myotonic disorder to be of central origin.

As was stated in my original paper, I regard the essential and characteristic symptoms of cerebellar disease, e. g. dyssynergia, dysmetria dysdiadokokinesis and intention tremor as manifestations of a disorder of the static system. The cerebellum is the essential organ for the control and regulation of the posture synergies of movement. This subject I have considered more in detail in a recent contribution to the symptomatology of the cerebellum.[4] There is also much in favor of the hypothesis that certain tonic postural spasms of cerebellar origin and the tonic rigidity of tetanus are essentially manifestations of the static system and the sarcoplasmic function of contractile tissue.

Static and Kinetic Seizures in Epilepsy.—In the motor manifestations of epilepsy there are also evidences of a static and kinetic mechanism.[20] The convulsive manifestations of epilepsy, both tonic and clonic, are related to the kinetic component of motility. This is true of both the jacksonian type and the general convulsion. The kinetic type of seizure is the common one and may result from a variety of causes, toxic, organic and emotional. In epilepsy, however, there is another type of seizure which I believe is related to the static system, and which, up to the present time, I have observed only in cases of idiopathic epilepsy.

Static seizures are characterized by a sudden loss of postural control, just as the kinetic seizure is characterized by a sudden release of motion mechanisms. Static seizures may occur alone, as a dissociated manifestation of epilepsy. The loss of postural control is sudden and shocklike, the patient falling to the ground with abrupt violence in response to the law of gravity. While the drop is sudden and immediate the postural relaxation is only of short duration, the patient rising almost immediately from the ground without assistance. The fall is usually associated with transitory loss of consciousness, which may, however, be very slight. In not a few instances there is scarcely any appreciable obscuration of consciousness. The fall is usually forward and is associated with a sudden relaxation or "giving way" of the lower extremities. As a rule there are no convulsive manifestations, although the two varieties of attacks may be combined. In the type of seizure just described the postural relaxation is more or less general in character, the patient falling in a heap from complete loss of postural control.

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20. Hunt, Ramsay: On the Occurrence of Static Seizures in Epilepsy (Trans. New York Neurol. Soc.), Arch. Neurol. & Psychiat. **8**:315 (Sept.) 1922.

The typical drop seizures are by no means common and when present they tend to recur with a certain degree of regularity and persistence. In addition to the general type of postural relaxation, these patients often show a more limited or local form of the disorder, which may be quite circumscribed in character and distribution, and is associated with myoclonic jerks or starts. Such myoclonic manifestations are not uncommon in the early morning hours in cases of epilepsy and particularly on arising, and are often relieved or lessened by the recumbent posture.

While in the present state of our knowledge one cannot assert positively that these myoclonic manifestations are related to the static system, it is my belief that a very close relationship exists, and that the myoclonic jerk is often only a compensatory kinetic manifestation in response to sudden localized relaxations in the posture sphere. Experimental evidence shows that some posture relaxation precedes, or accompanies, nearly every form of cortical movement, so that it is possible that both elements may play a rôle in these minor motor manifestations of epilepsy.

Under the title dyssynergia cerebellaris myoclonica I reported recently a group of cases with symptoms of cerebellar disease, associated with myoclonus epilepsy.[4] In these patients typical static or drop-seizures were observed and the question of the relation of myoclonus to the static system was considered. In one case histologic examination showed a primary atrophy of the efferent system of the cerebellum (cells of the dentate nucleus and the superior cerebellar peduncles). Such a lesion would cause a break in the static or posture system and may have some bearing on the myoclonia. In this group of cases it certainly is of more than passing interest that myoclonus-epilepsy with typical drop-seizures should be found associated with an organic lesion in the static mechanism.

I will now detail certain experimental evidence showing the importance of this system in the regulation of the static function of muscles.

Hering and Sherrington[21] in 1898, in their study of reciprocal innervation showed very clearly that postural inhibition and muscular contraction could both be elicited by electrical excitation of the cerebral cortex, and that there exists a form of coordinate innervation in which the relaxation of one group of muscles occurs as an accompaniment of the active contraction of another set. They mention the experimental

21. Hering and Sherrington: Ueber Hemmung der Contraktion willkürlichen Muskeln bei elektrisches Reizung der Grosshirnrinde, Pflüger's Arch. **67**:222, 1897; Inhibition of the Contraction of Voluntary Muscles by Electrical Excitation of the Cortex Cerebri, J. Physiol. **23**:, 1898-1899.

studies of Bubnoff and Heidenhain [22] on the excitation and inhibition of the motor centers of the cortex and similar studies of Exner on the cortex of the rabbit.

The experiments of Sherrington were carried out on cats and monkeys. He was able to show that stimulation of an appropriate center, e. g., that presiding over extension of the elbow, produced an immediate relaxation of the biceps, together with active contraction of the triceps. If the biceps or relaxing muscle is palpated during this experiment it becomes suddenly soft as if it were melting away under the examiner's touch, while the forearm is extended by contraction of the triceps. As soon as the stimulation is discontinued the arm returns to its previous posture of flexion. By weakening the faradic current, relaxation can in many instances be induced without any obvious contraction of the opposed muscles. The relaxation seems to occur quite synchronously with, or sometimes a little prior to, the contraction of the opposite group. The points of cerebral cortex from which relaxation and contraction of a particular muscle, e. g. biceps brachii, can be evoked respectively are distinct from one another, and often even in a small monkey lie more than a centimeter apart. In conclusion Sherrington made the following significant statement, "Besides therefore a localization for muscles according to their contraction there is also a cortical localization different in scheme and capable of demarcation by observations with relaxation as index. This correlation of relaxation of one set of muscles with contraction of the antagonistic set furnishes a further example of reciprocal innervation." One could hardly ask for more conclusive experimental evidence to prove the existence of motion and posture centers in the cerebral cortex.

Sherrington [23] also confirmed these results by cortical stimulation in decerebrate rigidity. After ablation of one cerebral hemisphere a homonymous extensor rigidity develops which presents an opportunity for examination of the sphere of excitation of the cortex on the extensor muscles of the crossed elbow and knee.

He found in the rolandic region of the monkey a cortical area, which gives markedly and forthwith, inhibition of the contraction of the extensors of the elbow, and another cortical area which similarly, when excited, inhibits the contraction of the extensor of the knee. This is in accord with his previous experiments with Hering already noted.

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22. Bubnoff and Heidenhain: Ueber Erregungs- und Hemmungs-vorgänge innerhalb der motorische Hirncentren, Pflüger's Arch. **26**:181.

23. Sherrington, C. S.: Decerebrate Rigidity and Reflex Coordination, J. Physiol. **22**:319, 1897-1898; Integrative Action of the Nervous System, New York: Charles Scribner's Sons, 1906; Decerebrate Rigidity, pp. 302-304; Reciprocal Inhibition, pp. 105-106.

Weed [24] in an elaborate study of decerebrate rigidity has still further clarified this subject. Weed found that inhibition of the extensor spasm of decerebrate rigidity could be obtained from the motor cortex of the rolandic area, from the mesial anterior portion of the internal capsule, from the mesial sixth of the crus cerebri.

Cobb, Bailey and Holtz [25] also investigated the genesis and inhibition of extensor rigidity in cats. They found that electrical stimulation of the cortex of the anterior lobe of the cerebellum produced an inhibition of the rigidity in the ipsolateral muscles. In agreement with Weed, they place the tentative pathway for this inhibition function in the anterior portion of the internal capsule, the crus, the contralateral portion of the pons, and anterior lobe of the cerebellum and the dentate rubral tract.

The Static and Kinetic Components in the Psychomotor Sphere.—In addition to the various somatic types of kinetic and static function a psychostatic and psychokinetic representation may be recognized in the psychic sphere.

Among the kinetic disturbances of psychic origin may be mentioned the psychic forms of tremor, chorea, convulsions, myoclonus, and convulsive tic. In the static sphere, are catalepsy, catatonia, astasia, stereotyped postures and attitudes. The symptoms of tonic perseveration which have been the subject of careful investigation by Wilson and Walshe [18] would also seem to reveal evidences of a dual representation in the psychomotor sphere in the so-called active and passive perseveration. The authors, in summing up, state that, "the repetition of a given movement in place of another and the continued repetition of a given movement when in a normal individual it would cease, should be known as active perseveration; whereas, the cessation of action which results in the maintenance of an attitude, either in the middle or the end of a given movement complex, should be known as passive perseveration." A somewhat analogous differentiation may be observed in the stereotyped movements and stereotyped postures of dementia precox, produced, I believe, by the release of kinetic or static representations in the psychic sphere.

Galvanometric Studies of Tonus and Reflexes.—In a recent study of tonus and reflexes, with the Einthoven string galvanometer, Wertheim-Salomonson [26] has furnished valuable clinical evidence of a kinetic and

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24. Weed, L. H.: Observations upon Decerebrate Rigidity, J. Physiol. **48**: 205, 1914.

25. Cobb, S., Bailey, A. A., and Holtz, P. R.: On the Genesis and Inhibition of Extensor Rigidity, Am. J. Physiol. **44**:239, 1917.

26. Wertheim-Salomonson, J. K. A.: Tonus and Reflexes, Brain, **43**:369, 1921.

static system. He has also confirmed the relation of these two systems to many of the disorders of motility, which I outlined in my original communication to The American Neurological Association. His investigations included the making of graphic records of the electrical and mechanical responses of human muscles in conditions of health and disease. He distinguishes two types of contraction, tetanic and tonic. The former occurs as a result of electrical or voluntary stimulation of the muscle; the tonic contraction, on the other hand, is secondary to the tetanic contraction. The tetanic contraction, which is the kinetic component, registers an electrical response on the string galvanometer; the tonic contraction produces no electrical response. For example, passive movements of the extremities produce no movements of the string galvanometer although there is a considerable "shortening and lengthening reaction" of muscle tone.

The convulsive manifestations of epilepsy, myoclonus epilepsy and chorea show a tetanic character of response with movements of the string. Clonus, tendon reflexes and cutaneous reflexes are also kinetic and cause an action current. Such disorders as catatonia, catalepsy and the carpopedal spasms of tetany are tonic, and produce no action current. It is interesting to note that postparalytic hypertonicity of muscles as in the spasticity of pyramidal tract disease and the rigidity of paralysis agitans are not associated with action currents and produce no movements of the string. In conclusion, he emphasizes the importance of this method in the study of motor disorders, and states that the absence of a typical action current is the surest means of differentiation between a tetanic and a tonic spasm.

Goodhart and Tilney [27] in their bradykinetic analysis of motor disorders by means of ultrarapid moving pictures have also confirmed my conception of the efferent system. Careful analyses were made of Huntington's chorea, intention tremor, dystonia musculorum deformans, and cases of cerebellar disease, from which they conclude that purposive movements depend on concurrent and synchronized operations of a kinetic and static mechanism.

II. THE VEGETATIVE NERVOUS SYSTEM

1. THE STATIC AND KINETIC COMPONENTS [28]

If the principle of a dual motion and posture function be applicable to all levels of the somatic system this should also apply to the vegetative mechanism, which represents the lowest phylogenetic level of the nervous

27. Goodhart, S. P., and Tilney, Frederick: Bradykinetic Analysis of Somatic Motor Disturbances, Neurol. Bull. **3**:295, 1921.

28. Preliminary report to the American Neurological Association, June 15, 1921.

system. If it be true of striped skeletal muscle, it should also be true of the unstriped visceral musculature, as one type of contractile tissue has merged imperceptibly into the other during the slow processes of evolution.

The object of the present chapter is to show that the vegetative nervous system has both static and kinetic mechanisms similar in function to those of the somatic nervous system. The static component is concerned with the postural function of involuntary muscle, and the kinetic component with motility proper. Evidence will be presented which favors the conception that the primitive postural function is subserved by the sympathetic system proper and that primitive motility is under the control of the parasympathetic system.

In this connection it is interesting to mention the conclusion of Marshall [20] after an exhaustive study of the muscles of both vertebrate and invertebrate forms. Marshall's investigations would appear to show that the disk mechanism of the striated muscle fiber is the final stage in evolution from the primitive intracellular network of the cell. This is represented in the unstriped muscle fiber by the longitudinal fibrillations. He thus traces from its origin the gradual development of the contractile mechanism of the muscle cell and shows its relation to the quality of movement. A movement is slow or quick, according to the quality and degree of differentiation of this contractile constituent of the muscle fiber.

All involuntary muscles which are innervated by the vegetative nervous system, with the exception of the heart, are of the unstriped variety, in this respect differing from the striated muscle of the cerebrospinal system.

The unstriped contractile fiber, like the striped variety, is also composed of two distinct substances, the fibrillae, a kind of primitive sarcostyle, and the sarcoplasm. In accordance with the general conception which I have formulated for the whole efferent system, it seems probable that the fibrillae of unstriped muscle are concerned with primitive types of motion (contractility), while the sarcoplasmic substance subserves the primitive types of posture (tonus).

2. ANATOMIC CONSIDERATIONS

The vegetative nervous system as it exists in man is a primordial mechanism and is the oldest portion of the nervous system in the phylogenetic sense. It corresponds to the ganglionic nervous system of the lowest forms of animal life and in man is concerned with the regula-

29. Marshall, C. F.: Observations on the Structure and Distribution of Striped and Unstriped Muscle in the Animal Kingdom and a Theory of Muscular Contraction, Quart. J. of Microsc. Sci. **28**:75, 1888.

tion of the automatic functions of the viscera, blood vessels and glands. Although under a certain degree of control from the higher levels of the nervous system, it is essentially an autonomic mechanism and was therefore termed the autonomic system by Langley.

According to the researches of Gaskell [30] and Langley,[31] the great pathfinders in this intricate realm, the vegetative nervous system may be divided into two parts or systems, which are anatomically, physiologically and pharmacologically distinct. One was termed by Gaskell the sympathetic outflow, which is represented by the thoracicolumbar portion of the sympathetic system which goes to make up the splanchnic, the cervical and sacral sympathetic. This portion of the vegetative or autonomic system is composed of non-medullated nerve fibers which pass to various groups of ganglions designated as vertebral, prevertebral and peripheral ganglions, because of their relative proximity to the vertebral column. The fibers of the sympathetic outflow originate in cells of the lateral horns of the spinal cord.

Gaskell's other system was termed the parasympathetic outflow, and differed from the preceding in that it emerged from certain portions only of the neuraxis, viz.: the gray matter of the midbrain, and medulla (cranial outflow) and the sacral region of the spinal cord (sacral outflow). Another important difference was that the fibers composing this system were medullated fibers and therefore of a higher order anatomically and functionally than were those of the sympathetic outflow. This midbrain, bulbar and sacral outflow of the parasympathetic system has been called the autonomic system by German writers, although this term was suggested by Langley for the whole vegetative nervous system. This is an abuse of Langley's original conception and only leads to confusion. I shall, therefore, use the terms sympathetic and parasympathetic to indicate these two divisions of the vegetative or autonomic nervous system.

(1) *The Sympathetic (Static) System.*—The fibers of the sympathetic system take their origin in the spinal cord (first dorsal to third lumbar) and pass by way of the anterior roots and white rami to the sympathetic ganglions which are ranged on either side of the spinal column as a distinct ganglionic chain (vertebral ganglions). From the vertebral ganglions the sympathetic fibers pass to prevertebral ganglions and thence to terminate in peripheral ganglion cells or ganglionic plexuses in connection with the organs themselves. The nerve fibers of the sympathetic are medullated as they take their exit from the spinal cord but lose their medullary sheath after leaving the vertebral

30. Gaskell, W. H.: The Involuntary Nervous System, New York: Longmans, Green & Co., 1916.

31. Langley, J. W.: The Autonomic Nervous System, Brain **26**:1, 1903.

ganglions. The preganglionic fibers (white ramus) are therefore medullated while the postganglionic fibers are non-medullated.

The cervical and sacral sympathetic may be regarded as white rami which have undergone a displacement and fusion in their respective regions to meet the requirements of the somatic innervation of the extremities.

All structures which are embraced under the general heading of glandular, visceral and vascular systems are innervated by the sympathetic outflow.

(2) *The Parasympathetic (Kinetic) System.*—The parasympathetic outflow emerges by three routes. The midbrain portion originates in the nuclear complex of the oculomotor nerve, passing to the ciliary ganglion, which it leaves via the short ciliary nerves to participate in the innervation of the iris, and the muscle of accommodation. The bulbar portion originates in the nuclear complex of the glossopharyngeal-vagus nerve. The latter, which is the so-called extended vagus, is the more important structure and participates in the innervation of the visceral contents of the thorax and abdomen.

The sacral portion of the parasympathetic, originates in the cells of the anterior cornua of the sacral part of the cord and emerges as the pelvic nerve. It participates in the innervation of the contents of the pelvis and lower abdomen. Because its function is similar to the vagus, this nerve is sometimes termed the sacral vagus. The fibers composing the craniosacral outflow of the parasympathetic system are medullated and pass directly to the mural and juxtamural ganglions of the peripheral visceral system, without passing through intermediate ganglionic structures as is the case with the sympathetic system. The fibers of the sympathetic system differ from those of the parasympathetic system in being non-medullated. They are of the type of the so-called fibers of Remak. The fibers of the parasympathetic system differ from those of the cerebrospinal nerves in being of smaller caliber with a fine medullated sheath. These morphologic differences may be regarded as representing evidence of phylogenetic development, an adaptation to the complexities of functional requirements.

All visceral, glandular and vascular structures are also supplied by the parasympathetic outflow, with the possible exception of the sweat glands, the pilomotor muscles of the skin and the blood vessels. There is, however, already some evidence to show that the vasodilators of the vascular system are innervated by parasympathetic fibers, and nearly all authorities, Higier,[32] Müller,[33] are agreed that the blood vessels, the

32. Higier, Heinrich: Vegetative Neurology, J. Nerv. & Ment. Dis. Monograph, Nos. 27 and 28, 1918-1919.

33. Müller, L. H.: Das Vegetative Nervensystem, Berlin: 1920.

pilomotor muscles and sweat glands have also an innervation of parasympathetic origin.

Therefore all visceral structures, with the possible exception of those noted above, have a double innervation, sympathetic and parasympathetic, which are mutually antagonistic in their function.

In the vegetative nervous system, therefore, two separate and distinct systems may be recognized which differ in structure and in function. They are mutually antagonistic and all vegetative structures are under this dual and antagonistic control. The cervicothoracic portion of the sympathetic is opposed by the cranial parasympathetic (midbrain and bulbar outflow) while the thoracicolumbar portion of the sympathetic is opposed by the sacral parasympathetic. Each group contains two classes of fibers: excitors and inhibitors, which control the various motor and secretory functions of the vegetative nervous system.

The Intrinsic Nervous Mechanism of Viscera: In addition to the sympathetic and parasympathetic outflow of the central nervous system which participate in the innervation of blood vessels, viscera and glands, there is also an important intrinsic nervous mechanism. This consists of a network of ganglion cells and fibers, situated within and on the walls of the viscera (mural or juxtamural systems). In its simplest form this is composed of nonmedullated fibers which represent a kind of diffuse nervous system, similar to that described by Bethe and Nicolai in the lowest forms of life, and which is still represented in the visceral nervous system of man.

In the gastro-intestinal tract of man there is also present a network of ganglions and fibers known as Auerbach's plexus, which represents a fairly complex neural mechanism. It is thought that this plexus plays an important rôle in the regulation of various automatic motor activities of the gastro-intestinal tract. For even if the stomach and intestines are completely separated from the sympathetic and parasympathetic systems, the motor functions still continue reflexly, by reason of the existence of this intrinsic ganglionic mechanism.

It is interesting to note that the vagus nerve of the parasympathetic system terminates in relation to the plexus of Auerbach, which is regarded by physiologists as having a motor function.

In the heart, this intrinsic nervous system has reached a much higher degree of differentiation and specialization, and practically controls the complicated automatic activities of this vital organ. There are aggregations of ganglions in the sinus, the auricles and the ventricles, all of which play an important rôle in the automatic function of the heart. The vagus nerve also terminates in relation to these ganglions. It is

interesting to note that the studies of Kuntz, Abel [34] and His, Jr.,[35] show that cells from the vagal nuclei migrate from the central nervous system to both the heart and gastro-intestinal tract; and that the cells of Auerbach's plexus are all of vagal origin. Physiologic experiments with drugs also confirm this embryologic evidence and would prove that vagal cells only are found on both the heart and the gut. This anatomic evidence would appear to favor the conception of the kinetic function of the parasympathetic outflow.

The existence of a well developed intrinsic nervous system explains in large part the automatic and autonomous nature of visceral functions which are maintained by the reflex activities of these primordial neural mechanisms.

In the gastro-intestinal tract, the plexus of Meissner is supposed to subserve a sensory function and probably plays an important rôle in primitive reflex movements and posture, an example of which is the myenteric reflex.

It is also very probable that the dual and antagonistic innervation of glands and viscera as represented by the sympathetic and parasympathetic system, is also present in this primitive intrinsic system.

3. *The Myostatic and Myokinetic Components of Involuntary Muscle.*—The contractile tissue which is under the control of the vegetative nervous system is composed of unstriped muscle fibers which are the forerunners of the striped muscle fibers of the sensorimotor system. The heart muscle occupies a place between the striped and unstriped muscle fiber and may be regarded as a transition form uniting these two fundamental types of contractile tissue.

The unstriped muscle cell is composed of two substances, fibrillary and sarcoplasmic, which may be regarded as the homologues of the sarcostyles and sarcoplasm of the striated muscle fiber. According to Heidenhain [36] and Apathy there are two distinct varieties of fibrillae in unstriped muscle and Benda [37] has confirmed this point of view.

There are finer fibrillae which are evenly distributed over the whole cross section of the fiber, and in addition, coarser fibrillae which form a concentric ring around the periphery of the fiber. These fibrillae which are supposed to represent different stages of development of the contractile fibrillary substance do not terminate in the ends of the cell

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34. Abel: The Development of the Autonomic Nerve Mechanism in the Alimentary Canal of the Chick, Proc. Roy. Soc. Edinburgh **30**: 1909-1910.

35. His, Junior: Die Entwickelung des Herznervensystems bei wirbelthieren Abh. d. k. Sachs. Gesellsch. d. wissensch., math. phys. Classe, **18,** 1891.

36. Heidenhain, M.: Die kontraktilen Substanz, Bardeleben's Handbuch der Anatomie des Menschen, Vol. 8.

37. Benda: Ueber den feineren Bau der glatten Muskelfasern der Menschen, Anat. Anz. **16** to **19**:214, 1902-1905.

but pass from one cell to another, thus forming a sort of continuity of fibrillar structure in a longitudinal direction. It is held by some histologists that unstriped muscle fibers form a kind of syncytial membrane, the fibrillae passing from one cell to another, and forming a kind of contractile network, Rouget [38] and McGill.[39]

Generally speaking the sarcoplasm is the predominating structure in unstriped muscle fiber and is in excess of the contractile fibrillae. This is in accord with the function of unstriped muscle which is characterized by sluggishness of movement and slow alterations of tone rather than by those sudden changes from activity to rest which characterize the function of skeletal muscles.

Another difference is the behavior of the muscle fiber after section of its nerve supply. In the case of striated muscle, the muscle fibers undergo atrophy. Section of the nerves passing to the visceral mechanism causes no atrophic changes in the unstriped muscle fibers. It must not be forgotten, however, that conditions are not wholly analogous, as after section of the extrinsic system (sympathetic and parasympathetic) of the vegetative mechanism there still remains the intrinsic nervous mechanism, which would be sufficient to maintain trophic function, if it were essential to this variety of muscle fiber.

Although the rate of contraction of unstriped muscle is very slow—much slower than that of the skeletal muscles—there are considerable variations in the rate of contraction in different organs and of the skeletal muscles themselves.

This is in accord with the statement which was previously made that the various types of movement and posture pass by imperceptible gradations, one into the other, and is also in accord with the evolutionary conception of contractile tissue.

The same is true of the histologic structures of the unstriped muscle fiber. There are many varieties of these cells which differ considerably in size, relative quantity of contractile fibrillae and sarcoplasm. In some unstriped cells the fibrillae have a spiral arrangement and others even show a tendency to transverse striation.

In the sensitive grading of contraction required by skeletal muscles, it is important that the fibers should act separately. In the smooth muscle of the heart the wave of contraction travels from fiber to fiber by virtue of the interlacing network of the fiber mass. It is likely that the lower form of motility of the other viscera is carried on in a similar manner.

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38. Rouget, C.: Mémoire sur les tissus contractiles et la contractilité, J. d. physiol. **6**:647, 1863.

39. McGill, Caroline: Histogenesis of Smooth Muscle, Intern. Mon. Anat. & Physiol. **24**:209, 1907-1908.

Unstriped muscle fibers also show certain peculiarities of function which are abolished or largely modified in skeletal muscles. Among the more important of these are: a tendency to automatic rhythmic contractility and tonicity, and a special sensitiveness to mechanical and thermal stimuli. This quality probably plays an important rôle in maintaining the automatic reflex activities of the viscera.

To summarize briefly, the essential point which I wish to emphasize as bearing on my conception of a duality of function of the efferent system is as follows: The kinetic component is represented in the parasympathetic, and the static component in the sympathetic outflow of the vegetative nervous system.

The fibrillary structure of the unstriped muscle cells is related to the kinetic fibers of the parasympathetic system, while the sarcoplasm is controlled by the static fibers of the sympathetic system. One is the *myokinetic* mechanism for the control of primitive forms of movement, in unstriped musculature, while the other is the *myostatic* mechanism for the maintenance of primitive forms of posture.

3. PHYSIOLOGIC CONSIDERATIONS

In the realm of physiology there are also many facts which corroborate the theory of a dual system of motility. Indeed, Mosso,[10] Botazzi,[40] Sherrington,[41] Magnus and Kleijn [42] and de Boer [43] laid the foundation for this conception by their work on the motor function of the vegetative and reflex nervous systems.

The unstriped musculature of the blood vessels, glands and viscera is arranged in layers, forming muscular sacs and tubes for the storage and conduction of fluid and semifluid contents. These muscular organs, undergo considerable alteration in volume and caliber as their contents are displaced by the force of gravity and muscular contraction.

According to the conception which was presented in the previous chapter the maintenance of static or postural tone is a function of the sarcoplasm which is under the control of the sympathetic system. The primitive motility is subserved by the contractile fibrillae which are under the control of the parasympathetic system. As this fibrillary structure of involuntary muscle forms an intercommunicating network or con-

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40. Botazzi, P.: The Oscillations of the Auricular Tonus in the Batrachian Heart, with a Theory of the Function of Sarcoplasm in Muscular Tissues, J. Physiol. **21**:1, 1897.

41. Sherrington, C. S.: Postural Activity of Muscle and Nerve, Brain **38**: 191, 1915.

42. Magnus and Kleijn: Die Abhängigkeit des Tonus der Extremitätenmuskeln von der Kopfstellung, Pflüger's Arch. f. d. ges. Physiol. **145**:455, 1912.

43. De Boer: Die Bedeutung der tonischen Innervation für die Funktion der quergestreiften Muskeln, Ztschr. f. Biol. **47**:239, 1914.

tractile membrane, a slow form of wavy movement is imparted to the muscular tissue of the viscera. The function of the sarcoplasm is to maintain the viscus in a state of postural tone in relation to its contents, analogous to postural fixation of skeletal muscles.

Where the viscus empties its contents into the narrow lumen of a tube or duct there is usually a thickening of the circular musculature of the region which is termed the sphincter and which holds the contents within the cavity. This sphincter function represents a specialization of the static or postural mechanism of the viscera and will be discussed later, under reciprocal innervation.

An original and very important contribution to the posturing function of viscera was made as early as 1882 by Mosso and Pellacani. These investigators showed that within certain limits the bladder adjusts its walls to its contents, maintaining the same degree of intravesical pressure, save for the pressure of the fluid, whether the organ be full or empty. They showed that the vesical bladder is no mere elastic sac which expands with increase of tension as it becomes distended, but a muscular membrane which adjusts itself in terms of postural tone to its contents. Sherrington [43] has pointed out the similarity of this manifestation in involuntary muscle to the plastic tonus observed by him in skeletal muscle and which underlies the so-called lengthening and shortening reaction of striated muscle.

Previous to this in 1871, Schatz [44] had already shown the existence of a similar mechanism in the uterus during parturition. He found by manometric measurements of intrauterine pressure that the column of mercury rose to 100 mm. during labor pains. In the pain free intervals, however, the column of mercury fell to 25 mm. in spite of the fact that the uterus had by contraction, changed its form and become smaller. Thus showing that in the uterus the same postural or capacity adjustment to changes of contents exist as in the case of the bladder.

Perhaps no tissue of the human body is subject to so many changes in size as unstriped muscle and this peculiarity is probably due to an actual displacement of the muscle cells. For example, Grützner [45] in his classical work on involuntary muscle has shown that the muscle cells of the empty stomach of a frog are arranged in superimposed layers, about twenty in number. When the frog's stomach is distended the layers of superimposed muscle fibers are reduced in number to four or five. This displacement of muscle cells takes place in both a longitudinal and transverse direction and is due to postural alterations in length without corresponding changes in tension. In this so-called "shortening and

44. Schatz, F.: Beiträge zur Physiologie der Geburtskunde, Arch. f. Gynaekol. **3**:58, 1871.

45. Grützner, P.: Die glatten Muskeln, Ergeb. der Physiol. **3**:12, 1904.

lengthening reaction" of muscle Grützner postulates the existence of an internal fixation or check mechanism, which he likens to a catch and ratchet mechanism, by means of which the contractile cell may become fixed in any length. In referring to the contracted stomach of a hungry frog he emphasizes the fact that this organ is not in a state of tetanus, analogous to the tetanic contraction of striated muscle, but that the muscle cells are simply resting in a state of shortened tone, a postural adjustment to diminished contents. In this relation he refers to the closing muscle of the molluscs and states that if this function were contractile and in the nature of tetanus it would give way under pressure. This particular phase of the subject has in recent years been the subject of a series of brilliant investigations by Von Uexküll.[46]

In conclusion Grützner states that smooth muscle is subject to the same changes as regards motion and posture as striped muscle, save that these activities are involuntary; furthermore, that the involuntary nervous mechanism which regulates these functions is automatic and reflex, and when it is apparently influenced by the voluntary nervous system, in reality it is only a voluntary production of a reflex action.

Von Uexküll's elaborate studies have contributed very materially to our knowledge of this important problem of muscle physiology. These researches were carried out on vertebrates and invertebrates and include both striated and non-striated muscle. His more important conclusions are briefly, as follows:

Formerly, in muscle physiology one considered only excitation, contraction and tonus, the latter being more apparent at rest. The conception of "Sperrung" which is the fixation or check component of muscular action has changed this. At the present time we must consider not only excitation and contraction, but, in the realm of tonus, also excitation and fixation. The contraction in indicated by the movement of the muscle mass. The fixation (Sperrung) is indicated by the hardness of the muscle.

This general conception of internal fixation was formulated by Grützner some years before for involuntary muscle, in which he postulated the existence of a catch and ratchet mechanism. Von Uexküll has shown that there are two kinds of fixation, one he terms maximal and the other gliding fixation (gleitende Sperrung). Maximal fixation is characteristic of smooth or non-striated muscles, while gliding fixation characterizes the postural function of striated muscle. Maximal fixation implies the utilization of the whole fixation power of the muscle.

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46. Von Uexküll: Die Physiol. des Seeigel Stechel, Zeitschr. f. Biol. **21**:73, 1900; with Noyon: Die Harte der Muskeln, ibid. **56**:139, 1911; Die Pilger Muschel, ibid. **58**:305, 1912; and with Gross: Studien über den Tonus (Die Schere des Fluss Rebses), ibid. **60**:334, 1913.

It has no gradations and is analogous to the "all or nothing" theory of Bowditch for the contractile function of unstriped muscle. Gliding fixation, on the other hand, implies the existence of various degrees and gradations of "Sperrung" corresponding to the varying shades and differences of muscular contraction which characterize the function of skeletal muscles. The skeletal muscles of man are capable of a weak or strong gliding fixation.

It is interesting to note that Parnas [47] has shown that the maximal fixation of unstriped muscle is unaccompanied by any demonstrable elevation of metabolism. This is similar to the results obtained by Pekelharing in striated muscle, who showed that the tonus or posturing function of muscle operates at a lesser turnover of energy than that of its contractile function. Therefore, the statotonic function of contractile tissue, which fixes the various parts of the body in postural adjustments, is a more economical process than the kinetic function, and is in harmony with the relative unfatigability of posture as contrasted with movement.

In this conception of internal fixation, Von Uexküll believes that the muscle fiber is converted into a mechanical apparatus by chemical means, by a conversion of sol into gel and a reconversion of gel to sol. The fixation fiber would therefore require two qualities, viz; those of clotting and unclotting the sarcoplasmic contents of the cell.

In the physiology of the heart a similar duality of function was noted by Botazzi [40] in 1897 in a study of the auricular tonus of the batrachian heart. This author reached the conclusion that the motor activity of the heart muscle consists of two components, one the beat which he refers to the fibrillary or disk system, and the other a tonic wave or oscillation which was thought to be of sarcoplasmic origin. After the demonstration by experiment of these two forms of muscular contraction, he formulated the suggestive theory of the function of sarcoplasm by which it is supposed to support and reinforce the quicker contractions of the sarcostyles or contractile mechanism.

Three years later, he investigated the action of the vagus and sympathetic nerves on the auricles of the tortoise, and confirmed his previous conclusions.[48] Stimulation of the vagus nerve abolished the elementary rhythmic contraction of the heart beat of the auricle, the tonus oscillations either increasing or remaining unaltered. Stimulation of the sympathetic, on the other hand, abolished the tonus oscillation and excited the elementary contractions or heart beat. He expresses the

47. Parnas, J.: Energetik glatter Muskeln, Pflüger's Arch. **134**:441, 1910.

48. Botazzi, P.: Action du vague et du sympathique sur oreillettes du coeur de l'emys europoea, Arch. Ital. d. Biol. **34**:17, 1900; and with Greenbaum: Function of Unstriped Muscle, J. Physiol. **24**:51, 1899.

opinion that these two types of muscle function have separate and distinct forms of innervation. The action of the vagus and sympathetic are therefore antagonistic and he believes that each nerve contains both excitomotor and inhibitory fibers, a subject which has been investigated in great detail by Spadolini.[49]

In the same year, 1900, Fano and Bodano[50] repeated the experiment of Botazzi on the auricle of the tortoise and reached the same conclusions as to the rôle of the sympathetic and vagus nerves and their relation to the tonus and contractile activities of the muscle, so that for heart muscle, which stands midway between striped and unstriped muscle, the duality of function, contractile and postural, has been demonstrated by exact experimental methods.

The tonus function or "lengthening and shortening reaction" of muscles of the peripheral static system was investigated by Sherrington, in 1909.[51] This intrinsic property of muscle tissue is dependent on postural tone, by means of which a muscle may be lengthened or shortened during contraction without corresponding changes of tension. This alteration in postural length without corresponding changes of tension facilitates the easy transition from posture to movement which characterizes motility and corresponds to what has already been demonstrated in visceral muscle by Schatz, Mosso and Pellacani and Grützner.

Sherrington in a more recent study,[41] (1915) of the postural activity of nerve and muscle has also shown the great importance of this posturing mechanism in the function of the hollow viscera and blood-vessels. He presents in most convincing manner the evidence in favor of a posture mechanism in both the vegetative and reflex nervous systems.

Sherrington has also recognized a duality of function in the various reflex activities of the segmental nervous system, and reached the conclusion that two types of reflex action may be recognized. One, the reflex movement, which he terms the phasic reflex, and the other a reflex posture which he terms the tonic or postural reflex; in the realm of reflex activity he has indicated the existence of a duality of function which I have extended to the whole efferent system.

Sherrington has also called attention to the fact that such phenomena as coordination and reciprocal innervation are as much a part of the

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49. Spadolini: Les actions antagonistiques dans les systèmes autonomes, Archiv. d. Fisiol. **15**:1, 1917.

50. Fano and Bodano: Sur des causes et signification des oscillations du tonus auriculaire dans le coeur de l'emys europoea, Arch. Ital. d. Biol. **34**:303, 1900.

51. Sherrington, C. S.: On Plastic Tonus and Proprioceptive Reflexes, Quart. J. Exper. Physiol. **2**:109, 1909; On Double Reciprocal Innervation, Folia neuro. biol. **111**, 1909-1910.

tonic or postural reflex as they are of the phasic reflex mechanism. His experiments on decerebrate rigidity, as well as those of Magnus and Kleijn, show conclusively that innervation and coordination are as important in the realm of posture as in that of movement.

Reflex action is therefore dependent on a peripheral kinetic system which terminates in the sarcostyles (myokinetic mechanism), and reflex posture is dependent on a peripheral static system which terminates in the sarcoplasm (myostatic mechanism). These are the neuromuscular representatives at the reflex or segmental level of the kinetic and static pathways of motility.

In any study of the physiology of motion of the vegetative mechanism, the automatic and autonomous nature of its function must be considered. This automaticity is so striking a characteristic of all visceral function, that some investigators believe that it resides in the inherent contractility of muscle tissue itself, and is quite independent of the nervous system. Others, however, while recognizing the inherent automatic and rhythmic qualities of involuntary contractile tissue insist that there must be an associated neural mechanism subserving the various reflex activities of vegetative function, e. g. the myenteric reflex, heart beat and vesical function; while the higher coordination of movement of the intestine is dependent on the sympathetic and parasympathetic outflow of the central nervous system. This reflex motor and postural activity of the gastro-intestinal tract is dependent on the intrinsic nervous mechanism (nerves and ganglions) of the heart, bladder, etc. which are under the control of the sympathetic and parasympathetic systems. That the peripheral vegetative system is autonomous is indicated by the relatively normal progression of digestion in the dog after transection of the vagus and the spinal cord. The so-called myenteric reflex, Cannon [52] or the law of the intestine, Bayliss and Starling [53] is a classical example of the function of this intrinsic mechanism. A rubber ball placed in the intestine gradually descends by the action of this reflex, i. e. contraction of the circular muscles above the bolus and dilatation below. It is a simple type of reciprocal innervation, the sensory components of which probably reside in the plexus of Meissner and the motor components in the plexus of Auerbach. That reciprocal innervation of higher order exists in the hollow viscera, is shown by stimulation of the parasympathetic system which causes relaxation of the sphincter muscle and contraction of the detrusor. On the other hand stimulation of the sympathetic causes contraction of the

52. Cannon, W. B.: Peristalsis, Segmentation and the Myenteric Reflex, Am. J. Physiol. **30**:67, 1912.

53. Bayliss and Starling: Movements and Innervation of the Large Intestine, J. Physiol. **26**:107, 1900; Movements and Innervation of the Small Intestine, ibid., p. 125.

sphincter and relaxation of the detrusor function. Gaskell and Langley have shown the existence of reciprocal innervation in the pyloric, vesical and rectal sphincters. Bainbridge and Dale [54] have also shown a similar reciprocal innervation in the gall bladder stimulation of the vagus causing contractions of the gall bladder and relaxation of the sphincters. The evidence shows that the extrinsic nervous system passing to the viscera is not in a position to produce a movement as such, but exercises this function by exciting or inhibiting the motor or postural activities of the intramural reflexes. All the evidence tends to show that the involuntary nervous system is built on the same plan as the voluntary nervous system. There are afferent and efferent pathways, excitation, inhibition and reciprocal innervation in both the autonomic and cerebrospinal systems.

Physiologic studies of the motor functions of the gastro-intestinal tract by Bayliss and Starling [53] also confirm the existence of a static and kinetic component in the splanchnic mechanism. These investigators have shown that the vagus is the motor nerve of the gastro-intestinal tract, while the sympathetic exercises a tonic and inhibitory function. Their enterographic studies show very clearly that, after stimulation of the vagus nerve, movement takes place in both the circular and longitudinal muscular layers of the intestines, while stimulation of the sympathetic produces a general increase of tone. These experiments would therefore seem to disprove the hypothesis of Basch and Fellner [55] of the Vienna School, who associated the longitudinal layer of the small and large intestine with the vagal and sacral outflow (parasympathetic) and the circular layer with the sympathetic system.

Langley and Anderson [56] have also shown that the large intestine receives its motor supply from the pelvic nerve (sacral parasympathetic) and that only the sphincter is innervated by the sympathetic, and Elliott has confirmed these results by use of the epinephrin method (cited by Gaskell).

Spadolini [49] in a comprehensive survey of the antagonistic action of the autonomic system and as a result of his own researches in this field has reached similar conclusions. According to his views the fundamental control of the contractile element of smooth muscle is the parasympathetic, while the tonic control is the sympathetic. The first system he believes has to do with relatively rapid movement, while the second

54. Bainbridge and Dale: The Contractile Mechanism of the Gall Bladder, J. Physiol. **33**:150, 1905.

55. Fellner, L.: Weitere Mittheillungen ueber die Bewegungs- und Hemmumgs-nerven des Rectums, Arch. f. Physiol. **56**:542, 1894.

56. Langley and Anderson: On the Innervation of the Pelvic and Adjoining Viscera, J. Physiol. **18**:, 1895.

produces a more general tonic response. He has also advanced the theory that both excitatory and inhibitory nerves of the sympathetic and parasympathetic pass together in their course and that weak stimulation inhibits function, while strong stimuli excites it.

In subsequent experiments he has been able to demonstrate these peculiarities in experiments on the stomach and intestines of the dog and cat.[57]

An antagonistic innervation of the vascular system has been shown to exist exactly as in the case of the viscera. It has been shown that stimulation of the hypogastric nerve (sympathetic) produces a constriction of the vessels of the internal and external genitalia. In contrast to this, however, stimulation of the nervus erigens (sacral parasympathetic) produces erection, i. e. dilatation of the corpora cavernosa. Again, stimulation of the cervical sympathetic produces constriction of the vessels of the submaxillary gland, while stimulation of the chorda tympani (midbrain parasympathetic) is followed by dilatation.

Gaskell states that there is also evidence to show that the constrictors and dilators of the pupil have an antagonistic action, similar in nature to that observed in the vegetative system. Waymouth Reid investigated this question by electrical tests and reached the conclusion that the law of reciprocal innervation holds for the eye as for other parts.

In this relation it is also interesting to record that the tensor tympani, which is the essential fixation or posturing mechanism of the tympanic membrane, is innervated by a filament from the sympathetic. The essential motor nerve of this region is the nervus stapedius, a branch from the facial nerve, which causes movement of the tympanum by its action on the stapes.

4. PHARMACOLOGICAL CONSIDERATIONS

In addition to the differences of structure and function which have been indicated, there are also pharmacologic evidences of a dual system of vegetative motility. The vegetative nervous system is essentially a reflex mechanism, the function of which is regulated by afferent stimuli, psychic stimuli and by the direct action of hormones.

These two systems, the sympathetic and parasympathetic, in which are represented the kinetic and static components of motility, react differently to certain drugs and hormones. In this respect there is a certain resemblance to the drug dissociations of the myokinetic and myostatic functions of the striated muscle fiber, strychnin acting on the contractile element of the muscle fiber, and veratrin on the sarcoplasm.

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57. Spadolini: Le azioni antagonistiche autonomi, Physiol. Abst. **3**:18, 1918-1919; Reazioni reflesse del tenue, ibid. **4**:23, 1919-1920.

These differences of reaction in the vegetative mechanism may be general or local. A drug or hormone may exercise a selective action on all portions of the system or the effect may have a very limited distribution. Certain drugs, for example, involve the central more than the peripheral portion of the mechanism.

Among the drugs which have a special action on the parasympathetic, pilocarpin is perhaps the most important. Physostigmin and muscarin also act specifically on the parasympathetic. In the realm of the sympathetic proper, epinephrin is the most important, and has the same effect as electrical stimulation. It is the sympathicotropic hormone formed by the cells of the chromaffin system and is the great chemical factor in maintaining tone.

The importance and special significance of epinephrin in the function of the sympathetic system is such that many investigators believe there must be a similar substance, also of endocrine origin, for the control of the parasympathetic. As yet, however, this hypothetical hormone has not been isolated.

It is very probable that the vegetative system is largely controlled by the endocrine glands and that hormones play a predominant rôle in the regulation of vegetative function, secretory, motor and inhibitory.

5. RELATION OF THE STATIC AND KINETIC COMPONENTS OF THE VEGETATIVE NERVOUS SYSTEM TO SYMPTOMATOLOGY

I shall now consider the relation of the vegetative system to symptomatology. While the evidence at this functional level of the nervous system is by no means so convincing as in the somatic sphere, there are nevertheless many facts in the autonomic system which are favorable to our hypothesis. Involuntary muscle, like voluntary, contains a contractile and a fixation mechanism which are innervated respectively by the parasympathetic and sympathetic systems. Langelaan, in his study of muscle tonus, reached the conclusion that contractile tonus is controlled by the cerebrospinal system and plastic or autonomic tonus by the sympathetic. He states that investigations on the tonus of smooth muscles have led to similar results. The plastic tonus of striped muscles, agrees in its essential features with the "Substanztonus" of P. Schultz, and the "neurogene tonus" of the same author resembles in many respects the contractile tonus of the skeletal muscles.

The postural system of visceral structures, like that subserving the function of skeletal muscles, is concerned with the maintenance of postural tone. This is the predominating function in hollow viscera and serves to adapt these organs to their contents and corresponding changes in hydrostatic pressure. Take, for example, the continuous postural activity of the vesical bladder and compare this with the comparatively slight degree of active motility which this organ manifests.

It may be stated, therefore as a general principle, that a disorder of the myostatic component of the vegetative mechanism causes a disturbance of postural tone, and a disturbance of the myokinetic component is characterized by a disorder of movement itself.

Such differences in function have already been noted by clinicians in the realm of gastric motility, and it is well recognized that increased tone and increased peristalsis do not always coincide. Observations are recorded in which there was a considerable diminution of the muscular tone in association with increased peristaltic activity.

Some years ago Eppinger and Hess [58] attempted to establish a specific symptomatology for the two great divisions of the vegetative nervous system, which had already been differentiated by the anatomic and physiologic investigations of Gaskell and Langley.

Largely by the use of pharmacologic methods, they separated into two large clinical groups, the rather complicated symptomatology of this region. One group, of parasympathetic origin, was termed vagotonia, because of the relation of the extended vagus to the symptomatology. The other, was referred to the sympathetic proper and was called sympathicotonia. Broadly speaking both syndromes were characterized by hyperexcitability of their respective systems. Furthermore, a state of hyperexcitability in one system was apparently associated with a state of hypoexcitability in the other. In other words, one or the other system assumed the center of the stage and dominated the clinical picture. This corresponds to the physiologic relationship of these two systems, which are mutually antagonistic and inhibitory.

In vagotonia, they recognized not only a condition of general vagotonia, but also local areas of vagotonic reaction, limited to the midbrain, bulbar or sacral distribution of the parasympathetic. As pilocarpin stimulates the parasympathetic, symptoms of vagotonia could be elicited or aggravated by the administration of this drug; and many of their deductions were based upon its effects.

Epinephrin, on the other hand, activates the sympathetic system and its administration would aggravate an already existing condition of sympathicotonia; or, under normal conditions, might induce symptoms of the sympathicotonic state. For these two important systems of the vegetative mechanism are antagonistic, and in conditions of disease are characterized by differences in symptomatology. As the systems are mutually antagonistic, an increase of tone in one is incompatible with an increase of tone in the other. Thus an individual who reacts unduly to pilocarpin shows a lessened reaction to epinephrin and atropin.

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58. Eppinger and Hess: Vagotonia, Monograph No. 20, J. Nerv. & Ment. Dis., 1915.

The symptomatology of vagotonia as given by these authors corresponds very closely to the experimental results of stimulation of the parasympathetic system, both by electricity and pilocarpin. Vagotonia in the sphere of the midbrain autonomic, is characterized by contraction of the pupil (myosis), spasm of accommodation and Von Graefe symptoms from excitation of the levator palpebrae. There also exists a tendency to salivation, lachrymation and sweating.

In the bulbar autonomic there is produced gastric hyperacidity, pylorospasm, cardiospasm and esophagospasm, spastic constipation, also diarrhea from increased secretion and increased peristalsis; spasm of the gall bladder and gall duct; bronchial asthma due to contraction of the muscle walls of the bronchi and increased secretion; bradycardia and mild anginoid symptoms from contraction of the coronary artery.

Vagotonia in the distribution of the sacral autonomic produces urgency of micturition, spermatorrhea, erections and rectal tenesmus. It is also interesting to note that all of the above symptoms of vagotonia are diminished by atropin and increased by pilocarpin. In conclusion they state that vagotonia is probably the expression of an inferior makeup, possibly associated with a defect of the chromaffin or of the thymic and lymphatic systems.

In sympathicotonia, there is a reversal of the pharmacologic reaction noted above. There is failure to react in the usual manner to pilocarpin, together with evidence of an undue susceptibility to epinephrin.

This conception of Eppinger and Hess is based on anatomic, physiologic and also to large extent on pharmacologic data. It represents an attempt to separate the function and symptomatology of the two great neural systems of the vegetative mechanism. It has by no means obtained general acceptance and is probably only in part correct. It is, however, a very suggestive hypothesis and has paved the way for further investigations of this important question. My reason for alluding to it at some length here is to show the importance of the parasympathetic outflow in the realm of active motility (kinesis), which to that extent tends to confirm my hypothesis of the dual nature of the efferent system.

In the realm of visceral symptomatology, a disorder of postural tone may be referred to the static component of motility. Among these may be mentioned: atony and dilatation of the stomach (gastrectasia), and intestines; atony and insufficiency of the sphincters, pyloric, cardiac, and rectal.

Corresponding disorders of the kinetic component are manifested in the gastro-intestinal tract by hyperkinesis, peristaltic unrest, nervous

vomiting and nervous diarrhea. It is also likely that spasmodic contractions of the sphincter muscles, pyloric, cardiac and rectal, are also of kinetic origin, although the possibility of spasmodic postural fixation must also be considered. The same may also be true of the so-called spastic form of constipation.

In the sphere of circulation, disorders of the purely contractile mechanism (heart beat) are referable to the myokinetic mechanism, e. g. tachycardia and bradycardia; while atonic states of the cardiac muscles (dilatation) are in part referable to the myostatic mechanism. Atonic states of the vascular system are also of static or postural origin and referable to the sympathetic system.

III. THE AFFERENT NERVOUS SYSTEM

1. THE STATIC AND KINETIC COMPONENTS[59]

In the foregoing chapters the evidence in favor of the functional and morphologic duality of the efferent system has been presented from various points of view. In the present chapter I shall consider the static and kinetic components of the afferent sphere.

The Kinesthetic and Statesthetic Systems.—The sense of movement and the sense of posture are well recognized components of deep sensibility. They are composed of sensory impressions derived from various sources, chiefly from the muscles, but also from the joints, tendons and fascia. The vestibular mechanism, is also closely related to the proprioceptive system and plays an important rôle in the regulation of postural tone. And it is interesting to note that recent investigators, Magnus and Kleijn,[42] Bárány[60] and Hunter[61] recognize the existence of a kinetic as well as a static labyrinth, in which, the semicircular canals yield kinetic impulses and the otoliths static impressions, in the regulation of movement and posture.

This point of view was foreshadowed by Sherrington,[23] some years ago in his classical work on the Integrative Action of the Nervous System; "In many forms of animals, e. g. in vertebrates, there lies in one of the leading segments a receptor organ (the labyrinth) derived from the exteroceptive field, but later recessed off from it; and this is combined in action with receptors of the proprioceptive field of the remaining segments. This receptive organ, like those of the proprio-

59. Preliminary report to the New York Neurological Society, June, 1922.

60. Bárány, R.: Diagnosis of Disease of the Otolith Apparatus, J. Laryngol. & Otol. **36**:229, 1921.

61. Hunter, R. T.: Disease of the Otolith Apparatus, with the Report of a Case, Arch. Neurol. & Psychiat. **7**:795, 1922.

ceptive field, is adapted to mechanical stimuli. It consists of two parts, both endowed with low receptive threshold and with refined selective differentiation. One part, the otolith organ, is adapted to react to changes in the incidence and degree of pressure exerted on its nerve-endings by a little weight of higher specific gravity than the fluid otherwise filling the organ. The other part, the semicircular canals, reacts to minute mass movements of fluid contained within it. These two parts constitute the labyrinth. The incidence and degree of pressure of the otoliths upon their receptive bed change with changes in the position of the segment, in which the labyrinth lies, relatively to the horizon line. Movements of the segment likewise stimulate the labyrinthine receptors through the inertia of the labyrinthine fluid and the otoliths."

More recently, Magnus and Kleijn [62] have investigated this subject. They rotated animals in a centrifuge at two thousand revolutions per minute, thus dislocating the otoliths in the otolith membranes. By testing the reflexes of the animals before and after rotation, and after examination of the otolith organs histologically, they reached the conclusion that these organs are exclusively concerned with the transmission of tonic labyrinthine reflexes to the body muscles, and in the maintenance of posture reflexes. They concluded also that the otolith apparatus is not stimulated by centrifugal force or, in the case of rectilinear motion, by any lagging of the otoliths on progression; and they call attention to the fact that, in case of the canals, the stimulus is on movement either starting or stopping; whereas, in the otolith membranes, the stimulus is constant from the time the head moves from the horizontal position. These investigators believe that, even in this position, there is a constant minimum stimulus for tonus. If this is the case, then disease of the otolith organs would cause signs induced by the position of the head rather than by rotation or movement of the head.

From their studies on the labyrinthine function of the rabbit, they recognize the existence of two types of reflex action; reflexes of movement and reflexes of posture. The former is concerned with the reaction of rotation and progressive movements; the latter with the tonic reflexes of skeletal muscles, and labyrinthine posture reflex and compensatory ocular fixation. Bárány [60] and Hunter [61] report cases in which postural anomalies, giddiness, and nystagmus were referable to disease of the otolith apparatus mechanism, the semicircular canals presenting no evidences of disease.

The trend of modern investigations, anatomic, physiologic and clinical seems, therefore, to indicate that the labyrinth has both a statesthetic and a kinesthetic function, separate and distinct from one another, subserving respectively the sensory aspects of posture and of motion.

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62. Magnus: Die Funktion der Otolithen, Ber. ü. d. ges. Physiol. **2**:174, 1920.

In the conception of a statesthetic and kinesthetic function, which is here presented, I have reference more particularly to muscle sensibility (myesthesia) and its relation to the dual functions of motility. For if it be true that the efferent nervous system from its earliest development in the vegetative mechanism to its highest expression in the cerebral cortex, shows evidence of a static and a kinetic mechanism, the existence of a similar division in the afferent sphere is very probable. For the efferent system is only one limb of the reflex arc, and where two separate physiologic systems exist subserving the function of motility, so different in the nature of their contractile function, there must also be corresponding differences in the function and morphology of their afferent mechanism.

One may postulate, therefore, in both skeletal and visceral muscle, the existence of special afferent systems for the transmission of sensations of movement and of posture to the central nervous mechanism. One is the kinesthetic component of muscle sensibility conveying impulses of movement (kinesthesia); the other is the statesthetic component conveying impulses underlying postural tone (statesthesia).

These two functions of contractile tissue, Sherrington [41] has described as follows: "Unstriped muscle like skeletal muscles, evidently functions for two main purposes, which in some ways it is possible and desirable to consider apart. Of these, one is the performance of movements which overcome resistance by the development of tension; the other is the adjustment of contractile length without necessary alterations of mechanical tension." This expresses what Von Uexküll [46] has demonstrated in both striated and nonstriated muscles; that muscle function may be resolved into two distinct components: a contractile component which is indicated by movement; and a fixation component which is manifested by the hardness of the muscle mass. In the muscle, for example, he has shown that these two functions are served by separate muscles and reflex mechanisms which may be isolated experimentally.

In the whole animal series, therefore, the contractile and fixation properties of the muscle fiber are independent functions. It has been suggested that the fixation mechanism of the muscle fiber is produced by the development of a mechanical apparatus through chemical means, i. e. by the conversion of sol into gel and its reconversion from gel to sol. According to this theory, clotting and unclotting of the sarcoplasmic contents of the cell is effected by means of a special innervation, which converts the muscle fiber into a static mechanism.

When one considers the high degree of differentiation of these two "effector mechanisms" their physiologic and anatomic differences, the

existence of special affectors and efferent systems which subserve these dissimilar and highly integrated types of reflex function can scarcely be denied.

Sherrington,[63, 23, 51] in a number of studies, has shown the great importance of the reflex factor in the production of reciprocal innervation. He has shown that the tonus of voluntary muscle may be inhibited by excitation of its antagonists. That movement at a joint in any normal direction involves the shortening of one set of muscles and the elongation of another (antagonistic) set. The stretching of a muscle produced by the contraction of its antagonist may excite (mechanically) the sensorial organs in the muscle that is under extension; in this way a reflex of pure muscular initiation may be started. One of his experiments shows "that electrical excitation of the central end of an exclusively muscular nerve produces inhibition of the tonus of its antagonist. (1) The central end of the severed hamstring nerve is faradized. This nerve contains in the cat about 1,810 sensory nerve fibers out of a total of 4,510 nerve fibers from the flexor muscles of the knee. The effect of this on the tonus of the extensor muscles of the knee is seen (*a*) in elongation of those muscles, (*b*) in temporary diminution of the kneejerk. (2) The exposed flexor muscles detached from the knee, and therefore incapable of mechanically affecting the position of the joint, are stretched or kneaded. This produces a reflex elongation of the extensor muscles of the knee and a temporary diminution of the kneejerk. The effects are in fact the same as those produced by faradization of the central end of the nerve supplying them. It may therefore be that reciprocal innervation, which I have pointed out previously is a common form of coordination of antagonistic muscles, is secured by a simple reflex mechanism, an important factor in its execution being the tendency for the action of a muscle to produce its own inhibition reflexly by mechanical stimulation of the sensory apparatus in its antagonist."

Sherrington,[23] in a discussion of reciprocal inhibition also states: "The striking correspondence observed between the reflex inhibition and the reflex contraction, when examined in one and the same type-reflex, allows the inference that the nerve fibers from the receptive

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63. Sherrington, C. S.: Further Experimental Note on the Correlation of Action of Antagonistic Muscles, Proc. Roy. Soc. of London, **53**:407, 1893; Experimental Note on the Movements of the Eye, J. Physiol. **17**:, No. 1, 1894; Cataleptoid Reflexes in the Monkey, Proc. Roy. Soc. of London **60**:411, 1896-1897; On Reciprocal Innervation of Antagonistic Muscles, ibid. **76**:160, 1905; On Reciprocal Innervation of Antagonistic Muscles, ibid. **79**:337, 1907; Proprioceptive Reflexes, ibid. **80**:552, 1908; On Double Reciprocal Innervation, Folia neuro-biol. **111**, 1909-1910.

field of the reflex each divide in the spinal cord into end-branches (e. g. collateral), one set of which, when the nerve fiber is active, produces excitation, while another set, when the nerve fiber is active, produces inhibition. The single afferent nerve fiber would therefore, in regard to one set of its terminal branches, be specifically excitor; and, in regard to another set of its central endings, be specifically inhibitory. It would, in this respect, be duplex centrally. There is analogy between the structural arrangement for reflex reciprocal innervation and that of Astacus claw, if it be supposed that the individual nerve fibers of the crayfish-claw preparation dichotomize, one division of the nerve fiber passing to the closing muscle, the other to the opening muscle; so that one division of the fiber exerts the excitor action, the other the well-known inhibitory, studied by Richet, Biedermann, Piotrowski, and others."

These experiments of Sherrington, I believe, also favor the inference that there are two separate systems in the afferent sphere for the control of posture and of movement; and that the harmonious cooperation of these two systems, in both the afferent and efferent sphere, underlie the phenomena of reciprocal innervation, by which the static or fixation system relaxes in harmony with the kinetic or contractile function of its antagonists. This harmonious cooperation of kinetic and static function, in terms of reciprocal innervation, is present in the cerebrospinal as well as the vegetative nervous system and is demonstrable in all forms of muscular activity, reflex, automatic-associated and isolated-synergic.

2. ANATOMIC CONSIDERATIONS

As has already been mentioned, the striated muscle fiber has two types of motor nerve endings, which are probably related to the dual systems of motility. One is the motor end plate which is the terminal of a medullated nerve fiber (myokinetic effector); the other is a sympathetic type of nerve ending, the terminal of a nonmedullated nerve fiber (myostatic effector). Both of these terminals are beneath the sarcolemma (hypolemmal) and therefore in direct relation with the contractile contents of the muscle fiber.

In addition to these motor types of nerve endings, the investigations of Huber [64] and Dogiel [65] have shown the existence of other terminals

64. Huber: Note on the Sensory Nerve Endings in the Extrinsic Eye Muscles of the Rabbit, Anat. Anz. **15**:335, 1898-1899.

65. Dogiel: Die Endigungen der sensiblen Nerven in den Augenmuskeln und deren Sehnen beim Menschen und den Saugethieren, Arch. f. mikroskop. Anat. **68**:501, 1906.

of a sensory character in relation to the muscle fiber. These are nerve endings of both medullated and nonmedullated nerves, and are situated outside the sarcolemma. They are found on the outer surface of the muscle fiber, the tendon and muscle-tendinous junction, as well as in the intermuscular connective tissue and are evidently sensory in their function.

Dogiel,[65] who used the methylene blue method and whose investigations were carried out on the ocular muscles (recti) of man and mammals, reached the conclusion that there are two kinds of sensory nerve endings for each muscle fiber. These are the terminals of both medullated and nonmedullated types of nerve fibers. In one form the nerve ending entwines the muscle fiber, very frequently throughout its whole length. The other, surrounds the end of the muscle fiber in the form of a palisade, the fiber fitting snugly into this end apparatus. Between these two typical types of sensory nerve endings of muscle fiber there are various transition forms. It is very evident from these investigations that muscle fibers are well supplied with sensory nerves, and nerve endings of both medullated and nonmedullated types.

It would be premature to attempt any correlation between these histologic studies and a possible kinesthetic and statesthetic function. As, however, in the effector sphere there is already considerable evidence showing that kinetic function is controlled by a medullated nerve fiber and static function by a nonmedullated nerve fiber, it is possible that a similar morphologic difference and correlation may hold for the affectors and the afferent system. For the greatest evolution and highest differentiation of motility is in the kinetic sphere, and it is therefore possible that posture function, which is automatic and secondary, is subserved in both the afferent and efferent sphere by nerve fibers of primitive nonmedullated character.

Course and Termination.—The statesthetic and kinesthetic components of muscle sensibility unite with fibers from other structures subserving the sense of movement and of posture and pass together in the spinal cord, the brain stem and the thalamocortical pathway. In conditions of disease, because of their proximity, both components of the proprioceptive system are usually involved together. That is, disorders of the sense of posture are usually associated with loss of the sense of movement.

Within the spinal cord, the kinesthetic and statesthetic systems pass together in the columns of Goll and Burdach to the nuclei of the posterior column. From these nuclei, secondary pathways pass in the corpora restiformia to the cerebellum in the interest of postural function, while other fibers, both kinesthetic and statesthetic, are continued

in the brain stem to their secondary termination in the optic thalamus. The ventral and dorsal direct cerebellar tracts pass directly to the vermis cerebelli from their primary stations in the gray matter of the spinal cord.

From the optic thalamus, kinesthetic and statesthetic impulses are conveyed by its commisural system to the corpus striatum for the regulation of automatic-associated movement (paleokinesis). From the optic thalamus, the kinesthetic and statesthetic systems are then continued in their tertiary and final pathway to the parietal lobe of the cerebral cortex. From the terminal sensory mechanisms underlying posture and motion sensibility, kinesthetic and statesthetic impulses are reflected to the kinetic and static spheres of the cerebral cortex, i. e. the corticospinal system which subserves a neokinetic function, and the frontopontine tracts which regulate the neostatic functions of the cerebellum.

In addition, therefore, to those sensory structures which participate in the reflex postural and kinetic functions of the segmental nervous system, three great sensory stations representing posture-motion groupings may be recognized: in the myelencephalon (nuclei of Goll and Burdach); the diencephalon (optic thalamus); and the neo-encephalon (parietal lobe).

3. RELATION OF THE STATESTHETIC AND KINESTHETIC SYSTEM TO SYMPTOMATOLOGY

It may be stated as a general principle of symptomatology that a disorder of the kinesthetic system produces a loss of the sense of movement—a kinetic ataxia; and a disorder of the statesthetic system produces a loss of postural sensibility—a static ataxia. The statesthetic system is the sensory component underlying plastic tonus (statotonus), the "lengthening and shortening reactions" of muscles and other manifestations of postural tone, e. g. reflexes of posture. The kinesthetic system is the sensory component underlying the "twitch," the contractile tonus (kinetotonus)—and reflexes of movement.

These two functions of the proprioceptive system are usually involved together and frequently in the same degree. In some cases, e. g. in tabes, a more selective involvement may occur, causing ataxia, loss of tendon reflexes, with little or no loss of postural tone; on the other hand, there may be well marked hypotonia without ataxia or loss of tendon reflexes.

Involvement of the corpus restiforme, by cutting off statesthetic or postural stimuli to the cerebellum produces a static ataxia. This is a pure ataxic disorder due to loss of the postural synergy of the cere-

bellum and is not associated with other disturbances of deep sensibility. Kinetic ataxia is an incoordination of movement; static ataxia is an incoordination of posture.

IV. COMMENTS AND CONCLUSIONS

In the preceding chapters, I have presented in detail the evidence from many sources in favor of the dual nature of the efferent nervous system. The conception of a static and kinetic mechanism, functioning together in the interest of motion and posture, finds confirmation in many different fields of research—in histology, physiology, biology, chemistry, comparative anatomy and neurology.

I have attempted to lay the foundation for the hypothesis that these two components of motility present a parallelism of structure and function at all levels of the efferent mechanism. In other words, that the neural systems of motility, like those of sensibility, may be resolved into more than one component, subserving different types of function, corresponding to special adaptations of the organism to surrounding physical forces.

The animal body in its relation to the outer world is in a state of rest or in a state of motion, functions which are subserved by the efferent nervous system and its effector organs.

Movement always starts from posture and terminates in posture. Indeed, posture follows movement like a shadow, adding strength, stability and accuracy to movement itself. In this sense the postural function is secondary and the automatic companion of movement. When movement ceases it serves to convert the muscle into a resting or static mechanism, which resists the continuous forces of gravity and atmospheric pressure.

There is mutual cooperation and harmony of these two systems during muscular activity. A striking evidence of this cooperation is the reciprocal innervation, which is represented in the muscular activities of both the vegetative and the cerebrospinal nervous system.

While I have stated that the static system is concerned with posture and the kinetic system with motion, each system cooperates to some extent in both movement and posture. The static system contributes to the stability and accuracy of movement; the kinetic system plays a rôle in sustaining posture and attitude. It is the harmonious interplay of innervation, denervation and reciprocal innervation in the realm of these two systems, which underlies the various phenomena of motility.

Not only in the nervous system, but in the muscle fiber itself, there is evidence of a duality of structure and function. Movement is dependent on the fibrillary structure of the muscle fiber and postural

fixation on its sarcoplasm. The differentiation of these two substances may be followed through the various stages of muscle development in the evolution of the striated from the nonstriated muscle fiber.

The dual nature of the vegetative nervous system, is shown by the posturing function of viscera and those more primitive forms of motility which are detached from voluntary control.

In the splanchnic division of the efferent system, the static component preponderates and is subserved by the sympathetic system. The kinetic component is represented in the parasympathetic outflow. These two systems also show very clearly a cooperation of function in the realm of excitation, inhibition and reciprocal innervation.

In the cerebrospinal nervous system evidences of a dual function are present at all of the great functional levels, archeokinetic, paleokinetic and neokinetic, which subserve respectively the functions of reflex, automatic-associated and isolated-synergic types of movement. Here also are found the phenomena of reciprocal innervation and the mutual cooperation of static and kinetic systems.

The chief correlating center of the static system is the cerebellum, an organ which I believe is concerned with the regulation of posture synergies of motility. This organ has important connections with the cerebrum and subserves the higher functions of posture.

In the peripheral nervous system, the evidence would seem to show that the fibers subserving a static function are nonmedullated and those of kinetic function are medullated. Here is a morphologic similarity with the sympathetic and parasympathetic systems of the vegetative mechanism, and is in accord with the secondary and automatic rôle of posture in the realm of motility.

The anatomic description of the static and kinetic systems, which I have given is tentative only, and to be regarded as a starting point for further refinements of localization. And while I have described these two systems in rather a schematic manner, it is with the full realization of the incomplete and provisional nature of my outline.

I have also considered the question of the static and kinetic components of sensibility in the proprioceptive system. For if contractile tissue is developing along two distinct evolutionary lines, in the interest of kinesis and stasis respectively, there must be corresponding afferent systems subserving kinesthetic and statesthetic functions.

In the realm of symptomatology there is also much to favor the conception of the dual nature of the efferent system, both in the somatic and splanchnic sphere. A number of disorders may be referred to the kinetic system, while others are related to the static mechanism. In general, a disorder of movement implies a kinetic localization of

origin, and a disorder of posture a disturbance in the static sphere. While both systems participate in any motor disorder, it is often possible to indicate one or the other system as primarily at fault.

In conclusion, therefore, I postulate for the whole efferent system a duality of function and structure in the interest of motility: a kinetic component for the production of movement; and a static component for the maintenance of posture.

Critical Reviews

YELLOW SPINAL FLUID ITS ORIGIN AND SIGNIFICANCE*

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The finding of yellow spinal fluid on lumbar puncture can no longer be considered rare; a review of the literature indicates its presence in a great variety of conditions. From a report by Nammack [1] a yellow coloration is found in about 1.6 per cent. of spinal fluids. This percentage is based on nine years' work by the meningitis division of the New York Health Department. Of 5,801 fluids examined, only 96 showed a yellow color. Over 350 cases are reported in the literature in such conditions as new growths of the brain and cord, compression of the cord by neoplasm or deformities of the vertebrae, hemorrhage into the spinal canal, and inflammatory diseases of the cord and meninges. This will serve to point out that the yellow coloration is not pathognomonic of any one pathologic state, but is dependent on certain factors common to many. This phase of the subject will be taken up in more detail later.

Froin [2] is usually credited with being the first to have reported the finding of yellow spinal fluid, but he was preceded by Busch,[3] in 1897, who noted its occurrence in a case of sarcoma of the third and fourth ventricles, and by Schroeder,[4] in 1899, who likewise found it with a diffuse sarcoma of the brain and cord. However, they did not note the spontaneous massive coagulation that was so characteristic of the cases reported by Froin in 1903. He described the spinal fluids from three cases of organic nervous disease in which he found yellow color, increased number of lymphocytes, and marked and rapid coagulation. Since then his name has been associated with the syndrome: xanthochromia, massive coagulation, and increased cell count.

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* Read before the Milwaukee Neuro-Psychiatric Society, Milwaukee, Wis., Oct. 26, 1922.

1. Nammack, C. H.: The Significance of Yellow Spinal Fluid, Am. J. M. Sc. **159**:540 (April) 1920.

2. Froin, G.: Inflammations méningées avec réactions chromatique fibrineuse et cytologique du liquide céphalorachidien, Gaz. d. hôp., Paris **76**:1005, 1903.

3. Busch: Quoted by Leschke (Footnote 20).

4. Schroeder: Quoted by Leschke (Footnote 20).

In 1904, Babinski [5] reported this syndrome in a case of syphilitic pachymeningitis. He was followed by Cestan and Ravaut,[6] Donath,[7] Fornaca,[8] Kleineberger [9] and others. Sicard and Descomps,[10] in 1908, stated that the syndrome was caused only by meningitis, but this was refuted by Blanchetière and Lejonne [11] who stated it could occur in tumors as well and reported its occurrence in a tumor of the dura.

In 1912 Mestrezat,[12] in a detailed study of the spinal fluid, collected twenty-one cases of xanthochromia, and considered it as having a definite relationship to compression of the cord and the formation of a lumbar culdesac. The first cases reported in the American literature were those of Cooper [13] in 1910, and of Collins and Elsberg [14] in 1914; they occurred with an angiosarcoma of the cord and a giant tumor of the cauda equina, respectively.

In 1915, Mix [15] reviewed thirty-three cases in the literature and reported an additional case with cord tumor. He considered only those cases that showed the complete syndrome of Froin, and therefore did not include all cases of yellow spinal fluid. Since then, its presence has been described by many observers. Elsberg and Rochfort,[16] in ninety-two cases of cord tumors and other chronic surgical conditions of the spinal cord, noted yellow fluid in fourteen, ten of

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5. Babinski, J.: Méningite hemorragique fibrineuse; paraplégie spasmodique lombaire; treatment mercuriel; guérison, Bull. et. mém. Soc. méd. d. hôp. de Paris **20**:1803 (Oct. 23) 1903.

6. Cestan et Ravaut: Coagulation en masse et xanthochromie du liquide céphalorachidien dans un cas de pachyméningomyélite du cone terminale, Gaz. d. hôp., Paris **77**:985, 1904.

7. Donath, J.: Beitrag zur Landry'schen Paralyse, Wien klin. Wchnschr. **18**:1327, 1905.

8. Fornaca: Coagulabilita e xantocromia del liquido cerebrospinale in un caso di lesione della coda equina, Gazz. d. osp. **27**:497 (Aug. 4) 1906.

9. Kleineberger: Ein eigenthümlicher Liquorbefund bei Rückenmarkstumoren, Monatschr. f. Psychiat. u. Neurol. **28**:346, 1910.

10. Sicard et Descomps: Syndrome de coagulation massive, de xanthochromie, et d'hémato-lymphocytose du liquide céphalorachidien, Gaz. d. hôp., Paris **81**:1431 (Oct. 20) 1908.

11. Blanchetière et Lejonne: Syndrome de coagulation massive et de xanthochromie du liquide céphalorachidien dans un cas de sarcome de la duremère, Gaz. des hôp., Paris **82**:1303 (Sept. 14) 1909.

12. Mestrezat: Le liquide céphalorachidien, Paris, 1912.

13. Cooper, C. M.: Cerebrospinal Fluid of Anomalous Character in a Case of Intraspinal Tumor, J. A. M. A. **55**:2298, 1910.

14. Collins and Elsberg: Giant Tumors of Conus and Cauda Equina, Am. J. M. Sc. **147**:493, 1914.

15. Mix, C. L.: Spontaneous Massive Coagulation of Cerebrospinal Fluid with Xanthochromia, Murphy's Surg. Clin. **4**:317, 1915.

16. Elsberg, C. A., and Rochfort, E. L.: Xanthochromia and Other Changes in Cerebrospinal Fluid, J. A. M. A. **68**:1802 (June 16) 1917.

which were extramedullary tumors, two gummas, one varicose veins of the cord, and one neuritis of the cauda equina. The same year Sprunt and Walker [17] reported 100 cases from the literature and added five of their own. More recently Nammack [1] reported ninety-six cases. Sixty of these, in which the diagnosis was confirmed, were tabulated and showed its occurrence in forty cases of tuberculous meningitis and in thirteen cases of poliomyelitis. In the past year reviews have been made by Greenfield [18] in England, by Lantuéjoul [19] in France, and by Leschke [20] in Germany who was able to collect 310 cases. This is the largest number collected together thus far. His figures are given in Table 1 and will be considered in comparison with the cases I have collected from the literature, which are shown in detail in Table 2.

TABLE 1.—*Cases Collected by Leschke*

	Cases	Cases Showing Coagulation
Bleeding from puncture	5	0
Trauma	30	1
Cerebral hemorrhage	11	1
Brain tumor	7	0
Cord tumor	67	16
Diffuse sarcoma of meninges	8	2
Tuberculosis of spine	20	16
Hemorrhagic pachymeningitis and leptomeningitis	7	2
Glioma	1	0
Tuberculous meningitis	65	12
Cerebrospinal meningitis and sequelae	10	3
Syphilitic meningomyelitis	6	2
Myelitis and meningomyelitis	10	0
Poliomyelitis	18	5
Myelitis transversa	3	1
Myelitis funicularis	1	0
Landry's paralysis	3	3
Cerebrospinal syphilis	12	0
Encephalitis lethargica	2	0
Encephalitis hemorrhagica after salvarsan	1	1
Encephalitis in eclampsia	1	0
Encephalitis in epilepsy	3	0
Localized recurrences of syphilis (Nenrorezidive)	2	0
Heart insufficiency	10	0
Pneumonia, appendicitis, malaria	7	0
Total	310	65

In Table 2 are grouped 339 cases of yellow spinal fluid from the literature. It is not claimed that the list is complete and includes all cases reported. In some instances the articles, or abstracts of them, were not readily available and these are omitted. In this class are the

17. Sprunt, T. P., and Walker, J. E.: The Significance of Xanthochromia of the Cerebrospinal Fluid, Bull. Johns Hopkins Hosp. **28**:80 (Feb.) 1917.

18. Greenfield, J. G.: On Froin's Syndrome and Its Relation to Allied Conditions in the Cerebrospinal Fluid, J. Neurol. and Psychopath. **2**:105 (Aug.) 1921.

19. Lantuéjoul, P.: Le Coagulation massive et spontanée du liquide céphalorachidien, Rev. neurol. **36**:339, 1920.

20. Leschke, E.: Ueber die Gelbfärbung der Cerebrospinalflüssigkeit, Deutsch. med. Wchnschr. **47**:376 (April 7) 1921.

articles by Heilig,[21] Pilotti,[22] and Lantuéjoul.[19] Besides these, other reports dealing with this subject have probably been overlooked, particularly in the foreign journals.

The reports of Nonne[23] and Raven[24] have given some difficulty. Greater attention was given by these observers to the increase of protein indicated by the phase I reaction, the yellow color or massive coagulation being noted only incidentally. Some writers have included all of Raven's series of forty-seven cases as having yellow colored fluids, but this is not correct. As far as I have been able to ascertain, only seventeen showed yellow fluids, eight with extradural compression and eleven with intradural compression. In Table 2, I have included only one of these, a case of tuberculosis of the spine. This leaves sixteen more to be added and brings the total to 355 cases.

In comparing the tables it will be noted that in certain pathologic conditions, Leschke has found a greater number of cases than I have, and in other conditions I have recorded the greater number. This can be seen more readily in Table 3. From this table it is apparent that Leschke has found 110 cases additional to those that I have included in my table. He has probably included the cases reported by Raven[24] which I have omitted. But even if these sixteen cases are deducted there are still ninety-four cases in excess. Even allowing for duplications this would carry the number of reported cases well over 400.

Another interesting comparison is in the number of cases showing the complete syndrome of Froin. Leschke found it in one-fifth of the cases he reviewed, while I found it in a greater number, nearly one-third of the cases presenting this feature. The percentage is probably somewhere between these figures. The difference may be due to the standard or dividing line of what constituted massive coagulation.

ORIGIN OF THE YELLOW COLOR

There has been considerable question and controversy as to the origin of the yellow coloration of the spinal fluid in these cases. Its

21. Heilig: Ueber Pigmenterythrozytose der Cerebrospinalflüssigkeit, Monatschr. f. Psychiat. u. Neurol. **29**:95, 1911.

22. Pilotti, G.: Sulla sindrome "xanthochromia, coagulazione del liquor," Rev. sper. di freniat. **44**:513, 1920.

23. Nonne: Ueber das Vorkommen von starker Phase I Reaktion bei fehlender Lymphocytose bei 6 Fällen von Rückenmarkstumor, Deutsch. Ztschr. f. Nervenh. **40**:161, 1910; Weitere Erfahrungen zum Kapitel der Diagnose von komprimierenden Rückenmarkstumoren, ibid. **47**:436, 1913.

24. Raven, W.: Die Bedeutung der isolierten Eiweissvermehung und der Xanthochromie im Liquor cerebrospinalis für die Diagnose von Kompression der Rückenmark, Deutsch. Ztschr. f. Nervenh. **44**:380, 1912; Weitere Beiträge zur Kenntnis des Kompressions-syndroms im Liquor cerebrospinalis, ibid. **49**: 36, 1913.

TABLE 2.—*Cases Collected from the Literature*

	Cases		Cases Showing Coagulation	
Inflammations:				
Tuberculous meningitis	51		7	
Epidemic meningitis	38		2	
Poliomyelitis	16		3	
Staphylococcus meningitis	2		1	
Meningomyelitis and pachymeningitis	13		11	
Landry's paralysis	4		4	
Meningomyelitis after stovain	1		1	
Meningoencephalitis	2		1	
Cerebrospinal syphilis	2		0	
Syphilitic pachymeningitis	11		7	
Neuritis of cauda equina	1		0	
		141		37
Inflammations with Pressure:				
Solitary tubercle of cord	1		1	
Myelitis funicularis	1		0	
Cystic disease	3		2	
Extradural abscess	5		5	
Gumma of cord	3		0	
		13		8
Extradural Pressure:				
Carcinoma of vertebrae	3		2	
Sarcoma of vertebrae	3		2	
Tuberculosis of spine	12		2	
Cholesteatoma	1		0	
Spondylitis	1		0	
Echinococcus cyst	1		0	
Fracture of spine	1		0	
Dislocation of vertebrae	1		1	
Cysticercus	1		0	
		24		7
Intradural Pressure:				
Tumor of cord and meninges—intramedullary	10		4	
extramedullary	35		18	
Tumor of cauda equina	10		9	
Syringomyelia	3		0	
Varicose veins of cord	2		1	
		60		32
Hemorrhage:				
Brain tumor	13		0	
Cerebral gumma	1		0	
Pachymeningitis hemorrhagica interna	2		0	
Meningeal hemorrhage	2		2	
Cerebral hemorrhage	5		0	
After puncture	3		0	
		26		2
Non-nervous Affections:				
Heart insufficiency	10		0	
Pneumonia, appendicitis	5		0	
Jaundice	1		0	
		16		0
Unclassified		59		22
Totals		339		108

TABLE 3.—*Comparison of Figures in Tables 1 and 2*

	Cases Table 1	Cases Table 2	Difference
Tuberculous meningitis	65	51	14
Cerebrospinal syphilis	12	2	10
Poliomyelitis	18	16	2
Cord tumors, Sarcoma of meninges	75	55	20
Tuberculosis of spine	20	12	8
Trauma, Fracture of spine	30	1	29
Cerebral hemorrhage	11	5	6
Hemorrhagic pachymeningitis	7	2	5
After puncture	5	3	2
Pneumonia, appendicitis, etc.	7	5	2
Myelitis transversa, Encephalitis lethargica, Encephalitis after salvarsan, Encephalitis in eclampsia, Encephalitis in epilepsy, Neurorecidive	12	0	12
Total			110

causation is not always clear, and is apparently not the same in all the various pathologic states. It is generally agreed that the yellow pigment is ultimately of hematogenous origin. As to the exact method of its origin from the blood stream several theories have been advanced, and have been much discussed.

According to origin, the cases may be conveniently divided into three groups: (1) Those of serogenic origin, as from transudative processes. (2) Those of hemolytic origin, as from minute hemorrhages, the hemoglobin being broken up into bilirubin. (3) Those of icteroid origin, as in jaundice.

The first group includes those cases in which no red blood cells are observed and in which tests for hemoglobin are negative. Such fluids are usually found in compression of the spinal cord with obstruction to the spinal canal. Thus a portion of the subarachnoid space is cut off and forms a pocket or culdesac. Sicard and Descomps [10] first called attention to this fact, but Mestrezat [12] first pointed out its significance in the production of the xanthochromia. Since then the importance of the pouch formation has been agreed to by most observers. Recently Ayer [25] produced xanthochromic fluids experimentally by cord compression from paraffin injected into the spinal canal.

The obstruction may be produced by tumor or by meningeal adhesions. The degree of obstruction determines to a considerable extent the character of the fluid. Some question has arisen as to the exact method of the passage of the color material from the blood stream to the cavity, but it is usually considered to be a transudative process. There is diminished pressure in the culdesac, as connection with the spinal fluid above the compression has been cut; and at the site of compression the pial veins are dilated and make it easy for transudation to occur.

Sprunt and Walker [17] favor the transudative origin and state, "It is not necessary to presuppose minute hemorrhages into the meninges to explain the color of the fluid in which no red blood cells can be found, and in which tests for hemoglobin are negative. From the large amounts of globulin and fibrinogen present, it is evident that considerable transudation of blood plasma must have occurred. Normal plasma, somewhat concentrated in the thermostat at 37 C. for several days, more nearly approximates in color the bright yellow of these fluids than do dilute preparations of hemoglobin so treated." Mix [15] has pointed out that the fibrin separates out from these fluids much as blood clot behaves, the fibrin content of the fluids being very close to that of the blood. However, Greenfield [18] states that this approxima-

25. Ayer, J. B.: Cerebrospinal Fluid in Experimental Compression of the Spinal Cord, Arch. Neurol. & Psychiat. **2**:158 (Aug.) 1919.

tion is never so complete as to render it identical. Hanes,[26] also explains the color on the basis of a transudation of blood serum. He believes that pressure on the veins of the pia at the site of compression of the cord results in stasis of the blood stream and allows transudation to occur. The high fibrin and protein content and the presence of but few cells support this view; the fluid thus is analogous to a pleural exudate in venous compression.

It is possible that the stasis of the spinal fluid in the isolated pocket may be an important factor. Gordon[27] remarks that the pigments accumulate in greater concentration where there is stasis and a limited area for absorption, as in a closed sac. He believes that the spinal fluid in the sacculated portion of the subarachnoid space, being cut off from the choroid plexus through which it is filtered, reverts to a simple lymphoid material, yellow, coagulable, and cellular. He reports an illustrative case in which meningitis obstructed the foramen of the fourth ventricle and led to the production of internal hydrocephalus. The enlarged brain thus produced was pushed into the foramen magnum and caused a separation and sacculation of the spinal arachnoid space. The fluid of the ventricles was normal while the spinal fluid was yellow and coagulated on standing.

Babes[28] found yellow spinal fluid in patients suffering with a heart that was unable to make a complete systole, or a systole sufficient to expel the blood from the ventricle. He explains the occurrence of the yellow coloration in these cases by stagnation of the blood, with transudation of blood pigments through the blood vessels into the spinal fluid.

In some instances minute capillary hemorrhages have been claimed in addition to transudation of blood serum. Mestrezat, one of the early workers in this field, believed that, in addition to the effect of stasis, there was an alteration in the walls of the cavity, of infectious or toxic character, which so affected the vessels that transudation or small microscopic hemorrhages occurred. This would be true in cases of obstruction of the spinal canal by inflammatory meningeal adhesions, or in certain vascular tumors. However, in most instances of compression with the formation of a subarachnoid pocket, the yellow fluids can be accounted for by the transudation of blood serum.

The second group includes those in which the yellow color is due to the presence of red blood cells or to dissolved hemoglobin. It

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26. Hanes, F. M.: The Spinal Fluid Syndromes of Nonne and Froin and Their Diagnostic Significance, Am. J. M. Sc. **152**:66, 1916.

27. Gordon, A. H.: Internal Hydrocephalus with Xanthochromia of Spinal Fluid, Canad. M. A. J. **9**:1005, 1919.

28. Babes: La xanthochromie du liquide céphalorachidien chez les asystoliques, Compt. rend. Soc. de biol. **76**:313, 1914.

includes cases of inflammation, neoplasm or trauma. The color of the fluid is due to dissolved hemoglobin or its derivatives. Usually it occurs without demonstrable compression of the cord. Many cases are recorded in the literature that illustrate the occurrence of xanthochromia with conditions permitting a slow escape of blood into the spinal fluid. In 1904 Cestan and Revaut [6] reported a case of syphilitic pachymeningitis with yellow fluid which they considered to be due to a serofibrinous exudate and minute hemorrhages into the fluid. Babinski [5] also explained the color of the fluid in his case on this basis. Nonne believed that the yellow color was due to a mixture of blood pigments as a consequence of hemorrhage; and Leschke identified the pigments experimentally as bilirubin, formed from the hemoglobin by a ferment that was produced by the action of the red corpuscles on the cells of the spinal meninges.

Red blood cells have been frequently found in the fluids from meningeal inflammations such as tuberculous and epidemic meningitis, and poliomyelitis; in a considerable number of such cases yellow spinal fluid has been noted. Here the hemorrhagic tendency is credited with playing the important part in the production of the color. Wallgren [29] regarded these minute hemorrhages as the explanation for the yellow color in his cases of epidemic meningitis. Nammack noted yellow fluids in about two-thirds of his cases of tuberculous meningitis. Reich [30] described a case of tuberculous meningitis in which the fluid was bloody; on centrifugation it became a golden brown. The sediment contained many red cells and lymphocytes. At necropsy the hemorrhagic condition was revealed.

Vascular tumors of the cord may give rise to repeated hemorrhage with the production of xanthochromia. Such a case was described by Blanchetière and Lejonne.[11] They found a small vascular tumor of the dura compressing the seventh and eighth dorsal segments of the cord and containing many hemorrhagic areas. The fluid contained only a few cells but they considered that the yellow color was due to hemoglobin derivatives from the minute hemorrhages that occurred during the growth of the tumor.

Very significant and interesting are the cases of pachymeningitis hemorrhagica interna reported recently by Dunn.[31] The first lumbar puncture yielded a hemorrhagic spinal fluid; subsequent punctures gave

29. Wallgren: Xanthochromia in Cerebrospinal Fluid, Acta. Med. Scandinav. **53**:303 (May) 1920.

30. Reich, J.: Ueber Gelbfärbung der Cerebrospinalflüssigkeit, Mitt. a. d. Grenzgeb. d. Med. u. Chir. **25**:721, 1913.

31. Dunn, A. D.: Pachymeningitis Hemorrhagica Interna, A Study of Five Cases of Non-Traumatic Hemorrhagic Spinal Fluid, Am. J. M. Sc. **163**:819 (June) 1922.

fluids that progressed from a red to brown and then to a bright yellow color, without cells. This is definite proof of the hemolytic origin of the yellow color in inflammatory conditions with hemorrhage.

Xanthrochromia with red cells is not limited to the pathologic conditions of the cord and its meninges so far discussed. There are many cases which suggest that conditions in the cranial cavity may be responsible for the production of the red cells and yellow color. These fall in two groups: those in which the hemorrhage is rather marked and due to vascular rupture; and those in which the hemorrhage is small and continuous, as in tumors of the brain and dura.

In the first group are those cases where there has been rupture of the middle meningeal artery or fine vessels of the leptomeninges as a result of skull fracture. After the third day there appears a yellow tinge to the spinal fluid which becomes more marked as the hemoglobin is transformed into bilirubin; the color continues marked until about the fifteenth day when it disappears. Chatelin[32] has also noted a faint yellow tint in cases of concussion of the brain and believes this is due to small capillary hemorrhages which usually accompany this injury. In disease of the vessels, such as in syphilis, arteriosclerosis, and alcoholism, rupture may occur with bleeding into the ventricles or subdural space. In such cases the fluids resemble those that follow skull fracture. Longcope[33] reports such a case in advanced arteriosclerosis and arterial hypertension, with hemorrhage into the lateral ventricle.

The second group is made up of those tumors of the brain which are in contact with the dura or extend into the ventricles. Red blood cells are usually present in the fluid, though they may be completely dissolved and leave only the yellow color. Spontaneous coagulation of these fluids rarely, if ever, occurs. Such cases have been reported by Quincke[34] and Vincent.[35] Greenfield[36] also noted a yellow fluid with decaying red cells in a case of cerebral gumma. Sprunt and Walker, in their review of 100 cases from the literature, grouped these cases as exceptions to those that are explained on the basis of compression and obstruction, and regarded the yellow color as due to repeated small hemorrhages into the ventricles or the subarachnoid space. In support of this view they add a case of endothelioma of the dura in which the

32. Chatelin: The Cerebrospinal Fluid, in Nelson's Living Medicine, Thos. Nelson & Sons, N. Y. **6**:97, 1920.

33. Longcope, W. T.: Reported by Sprunt and Walker (Footnote 17).

34. Quincke: Zu Pathologie der Meningen, Deutsch. Ztschr. f. Nervenh. **40**:78, 1910.

35. Vincent, Cl.: Quoted by Greenfield (Footnote 24).

36. Greenfield: Quantitative Albumin Estimation of Cerebrospinal Fluid, Lancet **2**:685, 1912.

spinal fluid was yellow and contained a few crenated red cells. An instance of bleeding into the ventricle is reported by Horrax [37] in a boy of twelve with a tumor of the pineal gland. The spinal fluid was straw-colored and contained a large number of distintegrated red cells. A similar fluid was obtained from the ventricles. In this case it was found that the tumor had grown into the ventricles and allowed the escape of red cells into the fluid.

Because of the presence of erythrocytes in the spinal fluid in these cases, the term erythrochromia has been applied, and some observers have proposed placing them in a separate group. Hanes [26] has attached considerable importance to this condition and has given the following characteristics of erythrochromic fluids: (1) the color varies from shades of brownish red to dark amber and changes from day to day; that of xanthochromia is a lighter yellow and is more constant; (2) red blood cells or their shadows are seen and hemoglobin tests are positive; (3) the fibrin content is not high and the fluids do not coagulate massively.

He distinguishes this group sharply from the xanthochromias, and would place any yellow fluid containing red cells in this group. This finely drawn division is hardly justified on a pathologic basis. In many cases the yellow color arises by transudation of the blood serum; in others it is due to repeated hemorrhages. This would give an apparent basis for the division; but there are cases in which both factors are operating, as in vascular cord tumors and in pachymeningitis with capillary bleeding. Such a separation, therefore, would lead to unnecessary confusion. Both factors, transudation and hemorrhage, are important and either one may predominate in a given case.

Another interesting view was presented by Schnitzler [38] who claimed that the yellow coloration does not come from the blood pigments, but from color derivatives classed under the general group of luteins. An illustrative case was reported by Clarke and Lansdown [39] of an intramedullary tumor of the cord. However this does not discredit the evidence that the color has been found to be due to bilirubin.

The cases of icteroid origin can be dismissed very briefly. The occasional instances in which the spinal fluid is discolored in icteroid states are not generally included in the classification of xanthochromia.

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37. Horrax, G.: Studies on Pineal Gland, Arch. Int. Med. **17**:627 (May) 1916.

38. Schnitzler, J. G.: Klinische Beiträge zur Kenntnis der mit Muskelatrophien verlaufenden Formen von multipler Sklerose und chronischer Myelite, Ztschr. f. d. ges. Neurol. u. Psychiat. **12**:310, 1912.

39. Clarke and Lansdown: Intramedullary Tumor of Spinal Cord, Brit. M. J. **1**:1009 (May) 1914.

A case has been reported in detail by Levison.[40] It is possible that yellow spinal fluid would be found quite frequently if more spinal punctures were made in patients with jaundice.

After intraspinal injections of serum, some cases show a yellow tinge of the spinal fluid which persists for some time. These cases should not be considered as true instances of xanthochromia.

ASSOCIATION WITH OTHER CHANGES

The yellow coloration is generally associated with other changes such as spontaneous coagulation, increase of globulins, and variations in the cell count, which permit a further classification of yellow spinal fluids. These are important and merit consideration.

Spontaneous coagulation is due to the presence of a large amount of albumin and fibrinogen. It was first noted by Lépine [41] in a case of tuberculous meningitis. The coagulation may be so marked as to take place in the needle, usually it requires ten to fifteen minutes and may take several hours. Where it is marked, the tube containing the fluid may be inverted without spilling the contents. The amount of albumin may be very high. Mestrezat reported it as high as 4 per cent. and Denis and Ayer [42] as much as 2.01 gm. per 100 c.c.

For the production of sufficient albumin and fibrin to cause coagulation, it is necessary to have some obstructive lesion of the spinal canal that interrupts the flow of spinal fluid, and the formation of a pocket in which the fluid stagnates. Mestrezat and others have considered this important.

An increase in albumin is one of the earliest changes in compression of the cord, often without cell increase; this constitutes the albumin-cytological dissociation syndrome of Sicard and Foix.[43] The degree and duration of the compression determine the amount of albumin, and therefore the occurrence of spontaneous coagulation. When the pressure has been insufficient to cut off the spinal canal entirely, there occurs a fluid containing an abundance of globulin and albumin, which does not clot and may not contain cells. Such a fluid corresponds to the syndrome observed by Nonne in cases of cord tumor. Hanes considers Nonne's syndrome as only an early stage of the same process that gives Froin's syndrome. The fluid may be colorless early and the

40. Levison, L. A.: Xanthochromia with Report of Three Cases, Arch. Int. Med. **26**:459 (Oct.) 1920.

41. Lépine, J.: Le liquide céphalorachidien dans les processus méningés subaigus d'origine rheumatismale, Lyons méd. **101**:298 (Aug. 23) 1903.

42. Denis, W., and Ayer, J. B.: A Method for the Quantitative Determination of Protein in the Cerebrospinal Fluid, Arch. Int. Med. **26**:436 (Oct.) 1920.

43. Sicard, J. A., et Foix, Ch.: Dissociation albuminocytologique au cours des compressions rachidiens, Presse méd. **20**:1013 (Dec. 4) 1912.

xanthochromia appear later. Therefore, depending upon the duration and extent of the compression, there may be found all variations from a colorless fluid with increased globulin to one showing the complete syndrome of xanthochromia and massive coagulation. Raven [44] states that rapid severe compression of the cord leads to xanthochromia more often than does a slowly increasing pressure.

The association of massive coagulation with xanthochromia was noted by Elsberg and Rochfort [16] in seven of their fourteen cases of chronic spinal disease with yellow fluids. This is 50 per cent. and is rather a high percentage; but Sprunt and Walker state from their analysis of 100 cases that it occurs in the majority of cases showing yellow fluid. On the other hand Nammack found it in only six instances in ninety-six cases of meningitis. Referring to Table 2 of 339 cases, massive coagulation occurred in 108 cases or in about one-third. A somewhat smaller percentage was noted by Leschke (Table 1).

Certain pathologic conditions producing xanthochromia seem to bear an important relationship to the presence of coagulation. For convenience the cases in Table 2 are grouped in six classes according to the main causative factors.

With inflammatory conditions, 26 per cent. showed coagulation; in intradural compression it was present in 53 per cent. When the inflammatory condition was associated with compression, the percentage was increased. In hemorrhage coagulation was extremely rare. The two cases in which it was noted were in cases of meningeal hemorrhage reported by Nammack. No details were given, but it is possible that additional factors were present.

An increase in globulin is usually present. All of Nammack's cases of meningitis with yellow fluid showed a marked increase. Elsberg and Rochfort noted it in all but one of their cases, the exception being a case of neuritis of the cauda equina. Abt [45] is of the opinion that all fluids with xanthochromia have increased globulin, although the quantity may differ.

Variations in the cell count as a rule depend on the presence or absence of inflammatory reaction. If the pathologic process is associated with an inflammation of the meninges there will be an increase in the cells, either lymphocytes or leukocytes according to the type of the infection. Both syphilis and tuberculosis produce a lymphocytosis, while the purulent types of meningitis are associated with a leukocytic increase. It is generally held that an increase of cells indicates some reaction on the part of the meninges, and some writers have used

44. Raven, W.: Der Liquor cerebrospinalis bei Rückenmarkskompression, Deutsch. Ztschr. f. Nervenh. **67**:55, 1920.

45. Abt, I. A., and Tumpeer, I. H.: The Significance of Xanthochromia of the Cerebrospinal Fluid, Am. J. Dis. Child. **20**:153 (Sept.) 1920.

the cellular content of the fluid as a differential point between tumors (without cells) and inflammatory processes (with cells). In noninflammatory growths such as the smooth tumors, there is no change in the cell count; but in certain diffuse sarcomas and in the giant tumors of the cauda very high cell counts may be noted. Sprunt and Walker found that of fifty-one cases of tumor, seventeen showed a cell increase. Therefore the cell count has a limited differential value.

Various combinations of the above features have been described. The first, and probably the best known, is that of Froin which consists of xanthochromia, spontaneous massive coagulation, and an increase in the cell count. As has been shown, the conditions necessary for its development are the closed cavity, and changes in the vessels of the cavity wall such as allow transudation to occur. Wegeforth, Ayer and Essick [46] demonstrated this closed cavity by an intraspinal injection of ink followed by a cisterna puncture from which no ink was obtained. At necropsy a block was revealed in the spinal canal at the sixth dorsal level.

TABLE 4.—*Froin's Syndrome*

	No. of Cases	Cases Showing Coagulation	Percentage
Inflammation	141	37	26
Inflammation and Pressure	13	8	61
Intradural pressure	60	32	53
Extradural pressure	24	7	28
Hemorrhage	26	2	7
Non-nervous affections	16	0	0

In addition to the well marked cases of Froin's sydrome, a number are found that do not conform in all particulars. These make up the cas frustes reported by various observers. Nonne's syndrome is the best known of these, and will be considered with other incomplete syndromes:

1. Cases showing massive coagulation but absence of xanthochromia, as illustrated by Lépine's case of tuberculous meningitis.[41]

2. Cases showing an excess of protein but no cell increase. Yellow color may or may not be present. This is usually known as Nonne's syndrome. He reported a number of cases of cord tumor with these appearances. Raven also reviewed a number of such cases. The German observers were more interested in the large amounts of globulin found in the fluids (Nonne's syndrome), while the French observers looked for xanthochromia and massive coagulation (Froin's syndrome).

3. Cases showing yellow color, spontaneous coagulation but no cell increase. This a combination of Nonne's and Froin's syndromes, and occurs more frequently than the complete syndrome of Froin

46. Wegeforth, Ayer, and Essick: The Method of Obtaining Cerebrospinal Fluid by Puncture of the Cisterna Magna, Am. J. M. Sc. **157**:789, 1919.

with increased cells. Indeed most observers do not distinguish this from the syndrome described by Froin, and have included such cases under that classification without regard to the cell count. Blanchetière and Lejonne in 1909 were the first to call attention to the fact that few cells may be present in Froin's syndrome.

4. Cases showing yellow color and massive coagulation on some punctures and not on others. Sicard and Descomps report such a case and regard the variations in the findings as being due to friable, transitory adhesions.

5. Cases showing yellow fluid with little albumin, and red blood cells, but no clotting. Such cases were noted in meningeal and cerebral hemorrhage. These are the erythrochromias of Hanes.

6. Cases showing yellow color, increased cells and varying amounts of albumin, but no massive coagulation such as occurred in the cases of meningitis reported by Nammack.

SIGNIFICANCE OF XANTHOCHROMIA

All types of yellow spinal fluid are significant of organic disease, but not necessarily of the nervous system. However, with the few exceptions such as occur in jaundice it is indicative of organic nervous disease. In the cases of jaundice the diagnosis is obvious and the finding of yellow spinal fluid may be expected without changes being present in the nervous system.

The yellow coloration is not as important as other features that accompany it such as spontaneous coagulation and variations in the cell count. Indeed these features are often more characteristic and diagnostic than the yellow color. On the basis of coagulability of the fluid the cases may be divided into two groups.

The first group includes those in which there is a sufficient increase in albumin for spontaneous coagulation to take place. This finding is more pathognomonic than the xanthochromia itself. It practically always indicates an obstructive lesion of the spinal canal with interruption of the flow of the spinal fluid. Mix states that whenever spinal fluid is found to conform to this syndrome it means there is: a meningitis which has sealed the meninges to the surface of the cord; a pachymeningitis or tumor which compresses intradurally; or an extradural tumor. From his study of thirty-three cases of Froin's syndrome he stated that it occurred in three types of cases: (1) Certain acute cases probably all meningeal, and probably including Landry's paralysis. In these cases it would seem that there is an agglutination somewhere in the lower dorsal or upper lumbar portion of the spinal canal. As the meningitis subsides and the transitory agglutinations dissolve, the cerebrospinal fluid tends to return to normal. (2) Paraplegias of either flaccid or spastic type depending on whether the process is con-

fined to the cauda equina or to the lower part of the cord. In most of these cases there has been a pachymeningitis of the conus. (3) Tumors of two types. The first are neoplastic formations, especially the sarcomas and granulomas, which limit themselves to the dura and pia and divide the canal into two compartments, the lower one containing the yellow fluid. The second are those smooth tumors that apparently cut off the lower part of the canal by a ball valve action without adhesions.

Sprunt and Walker noted this syndrome in two cases of extramedullary tumor and Elsberg and Rochfort found it in ten out of twenty-four of their cases of extramedullary growths. Other cases have been reported by Mix [15] and by Parker.[47] It occurred regularly in the cases of giant tumor of the cauda equina. An instance of intramedullary tumor giving the syndrome was reported by Adams;[48] but it is more often found in the extramedullary growths. This agrees with Raven,[44] who states that spontaneous coagulation of spinal fluid has been seen principally in extramedullary tumors, intradural in origin.

Instances of Froin's syndrome in other conditions have been reported in syphilitic pachymeningitis [5, 6, 36] tuberculous meningitis,[1] poliomyelitis,[49, 1] solitary tubercle of the cord,[50] gumma of the cord,[16] and in tuberculosis of the spine.[51, 52]

It is doubtful if the complete syndrome of Froin ever occurs in conditions other than obstructive lesions of the spinal canal. Levison [40] believes that the few instances in which the syndrome has been reported in acute conditions such as tuberculous meningitis and poliomyelitis, may be accounted for on the assumption of an interference with the continuity of the spinal canal by an exudative process or an extravasation of blood.

The second group is made up of those cases in which spontaneous coagulation does not occur. Here the yellow color is significant of hemorrhage. It includes the cases of acute meningeal inflammation

47. Parker, H. L.: The Diagnosis of Tumors of the Cauda Equina, Conus, and Epiconus Medullaris, Am. J. M. Sc. **163**:342 (March) 1922.

48. Adams, D. S.: Glioma of Lumbar Cord, Boston M. & S. J. **186**:738 (June 1) 1922.

49. Abramson: The Spinal Fluid in Poliomyelitis and Its Differentiation from Fluids of Other Infections, Am. J. Dis. Child. **10**:344, 1915.

50. Thalhimer, W., and Hassin, G. B.: Clinicopathologic Notes on Solitary Tubercle of the Spinal Cord, J. Nerv. & Ment. Dis. **55**:161 (March) 1922.

51. Sicard, Foix et Salin: Reactions du liquide céphalorachidien au cours de la pachyméningitie Pottique, Presse méd. **18**:977 (Dec. 28) 1910.

52. Bromer, R. S.: The Syndrome of "Coagulation Massive et Xanthochromie" Occurring in a Case of Tuberculosis of the Cervical Spine, Am. J. M. Sc. **151**:378 (March) 1916.

such as were observed by Abramson [49] in poliomyelitis, Levison [40] in syphilitic pachymeningitis, Dunn [31] in pachymeningitis hemorrhagica interna, and by Nammack in tuberculous meningitis and epidemic meningitis. Here also are included the cases of brain tumor [37, 17] giving rise to repeated hemorrhages into the ventricles or subarachnoid space, as well as cases of more marked bleeding such as apoplexy [33, 20] and skull fracture. Spinal puncture alone does not indicate the type of lesion in these cases, and must be considered with the general physical findings. In these cases the cell count and the varieties of the cells are important. Yellow spinal fluid after skull fracture is significant of rupture of meningeal vessels and indicates a greater degree of injury.

Variations in the cell count have some significance; if the pathologic change is associated with inflammation of the meninges there will be an increased cell count, but when due to noninflammatory growths no increase is present. When the cellular elements are present in more than normal numbers, syphilis or tuberculosis should be suspected. In poliomyelitis and epidemic meningitis, the leukocytes are increased; in tuberculosis and syphilis there is a lymphocytosis. In giant tumors of the cauda equina, very high counts have been noted. Occasionally large cells are observed, but it is difficult to demonstrate the neoplastic character of isolated cells.

As to the localization of the lesion Raven [44] states that as we descend the cord the tendency to high protein content diminishes but the tendency to xanthochromia increases; it would therefore appear that yellow fluid indicates a lesion in the lower portion of the spinal canal. This is probably true in most instances of cord compression, for Elsberg and Rochfort found the lesion in the lower dorsal or lumbar region in all but one of their cases with yellow fluid.

Wallgren [20] is practically the only observer who has reported any data of prognostic significance in the yellow coloration of the fluid. He found the mortality in 103 cases of epidemic meningitis, after the first twenty-four hours, 19.6 per cent. In seventy-four cases without yellow color it was only 14.8 per cent.; in the cases with xanthochromia it was 39 per cent. He concluded that the gravity of the case was increased by xanthochromia and warned of the necessity for injecting serum into the ventricles.

Yellow spinal fluids have a certain diagnostic value in relation with other findings. Yellow spinal fluid that coagulates spontaneously justifies, in most instances, the suspicion of an extramedullary tumor, especially if the cell count is normal. If there is an increase in lymphocytes, tuberculosis or syphilis would also have to be considered. In a case with yellow spinal fluid that does not coagulate, one would suspect meningitis or hemorrhage. Cases with meningeal symptoms

would suggest the diagnosis of tuberculous meningitis, epidemic meningitis or poliomyelitis. In cases of hemorrhage there would most likely be the findings of arterial disease or of injury to the spine or the skull.

Mix states that if Froin's syndrome be present an operation is indicated, provided the case does not belong to the acute meningitis group, and syphilitic pachymeningitis can be excluded.

CONCLUSIONS

1. Xanthochromia has been reported in over 350 cases in the literature in association with pressure on the cord, hemorrhagic inflammation and hemorrhage into the spinal fluid.

2. The xanthochromia is ultimately of hematogenous origin, and may develop in two ways: transudation of blood serum; and hemorrhage. Both are important and one or the other may predominate in a given case.

3. Froin's syndrome, xanthochromia and massive coagulation, occurred in about one-third of the cases.

4. On the basis of spontaneous coagulation, yellow fluids are divided into two groups. As a rule, those showing coagulation indicate obstruction of the spinal canal; those without coagulation indicate hemorrhage into the spinal fluid.

5. Xanthochromia, with few exceptions is indicative of organic nervous disease.

6. An exact diagnosis of spinal disease cannot be made on the basis of the spinal fluid findings alone. In general: xanthochromia and massive coagulation indicate meningeal adhesions or compression of the cord by tumors; xanthochromia without coagulation indicates meningeal infllammation or hemorrhage into the spinal fluid.[53]

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53. Additional reference to articles of interest in the literature will appear in the reprints.

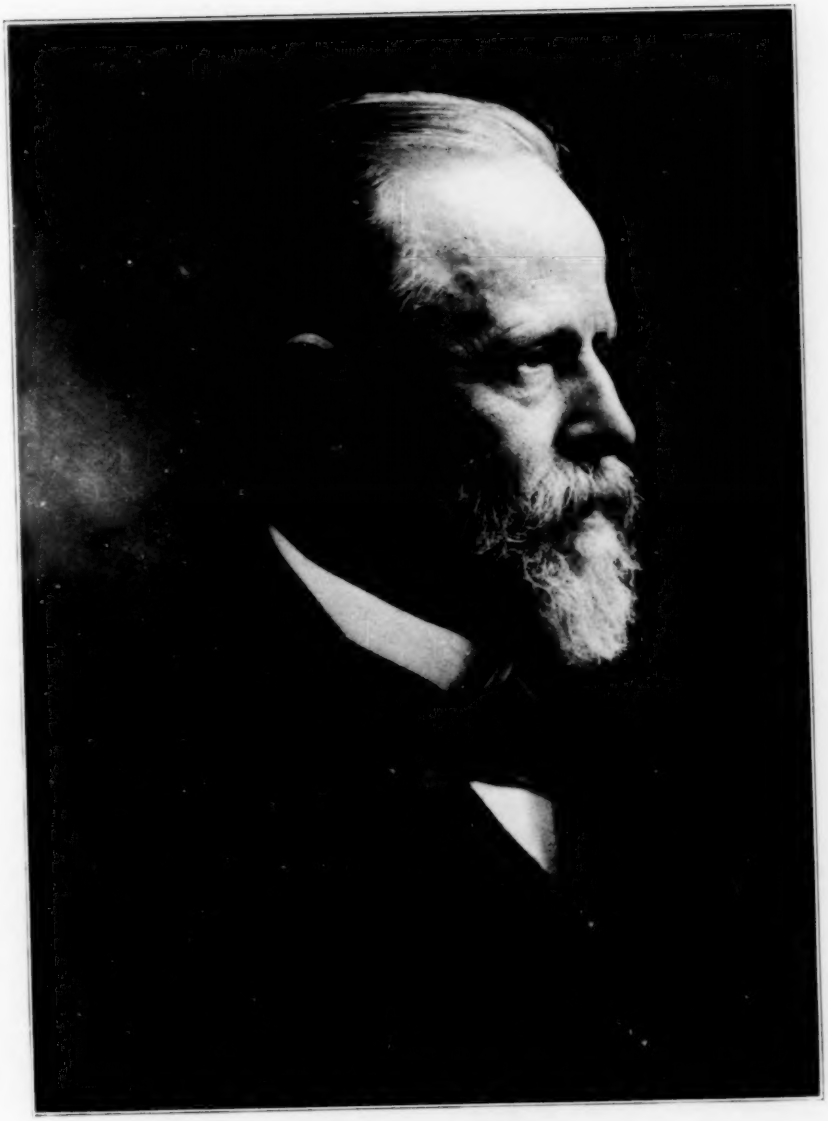
Obituary

HEINRICH OBERSTEINER

1847-1922

From the moment when Obersteiner entered on his career as a teacher in Vienna, students began to come from America, attracted by his personal charm, his ability as a teacher and his excellent command of English. It was soon recognized that Obersteiner was more than a good teacher, and that he belonged with the great Viennese scholars of tradition in whom were combined excellence of scientific attainment with exalted love for humanity.

Obersteiner was born, Nov. 13, 1847, and died, Nov. 19, 1922. As the son of a physician, he was familiar with sick people from his earliest youth. His instructors in medicine included Hyrtl, Brücke, Skoda, Oppolzer and Billroth, men who had done much to establish the fame of the medical school of Vienna. Obersteiner did not simply succeed them; he also inherited from them the mantle of creative genius. Through his parents he became the owner of a private sanatorium for mental diseases, and his energies thus received twofold direction: on the one hand toward practical neuropsychiatry, on the other toward the academic study of the anatomy, physiology and pathology of the nervous system. He was one of the first to attack the problem of general paralysis and discovered its relations with syphilis. In collaboration with his pupil Redlich, he demonstrated that tabes dorsalis is a disease of the posterior roots. He produced epilepsy experimentally and proved that it had a relation with heredity and trauma. He was the first to investigate commotio spinalis and proved that it is caused by molecular changes in the nervous tissue. He was the first to describe allochiria. His studies on morphinism, the intoxication psychoses and hallucinations, together with his book on general paralysis, assured him lasting fame as a clinician. As an anatomist he devoted himself to the study of the normal histology, comparative anatomy and pathology of the cerebellum. He was the first to establish that the pigment in ganglion cells is a lipochrome and a product of catabolism. He found this pigment also in the glia cells, and from his investigations it seems not improbable that corpora amylacea are derived from glia cells. In the anatomy of nerve tracts, he occupied himself with the association paths and with abnormal fasciculi. But he is known best by his text book, the first edition of which appeared in 1888; the fifth German edition was published in 1912, and has been translated into all modern



HEINRICH OBERSTEINER

languages. It is today probably the most comprehensive text book on the theory of neurology, simple and lucid, with careful attention to detail. The Neurologic Institute of Vienna, probably the first of its kind, founded in 1882, will stand as his lasting monument. It already has given an incomparable training to a great many pupils and has produced more than five hundred scientific publications, most of them in the "Arbeiten aus dem Wiener neurologischen Institute."

Obersteiner was admitted to the faculty in 1873; later he became associate professor, and finally the supreme rank of Professor with the title "Hofrat" was conferred on him. Honors were showered on him from all parts of the world, none more greatly appreciated than the love and esteem of his pupils to whom he devoted himself with invariable courtesy and good will. As a scholar of international repute he had scarcely a living equal. There is no field of anatomic, clinical or therapeutic neurology that has not been enriched by his studies. As a teacher he reached the highest pinnacle of success and became one of the foremost exponents of neurology. As a man he won the love and admiration of all who had the good fortune to come in contact with him.

OTTO MARBURG, VIENNA.

News and Comment

FIFTIETH ANNUAL MEETING OF THE AMERICAN NEUROLOGICAL ASSOCIATION

The fiftieth annual meeting of the American Neurological Association will be held in Philadelphia, in 1924, with the following officers: president, Dr. Charles K. Mills, Philadelphia; vice-presidents, Dr. Charles L. Dana, New York, Dr. Morton Prince, Boston; secretary-treasurer, Dr. Frederick Tilney, New York.

Abstracts from Current Literature

THE GANGLIA, PLEXUSES, AND NERVE-TERMINATIONS OF THE MAMMALIAN LUNG AND PLEURA PULMONALIS. O. LARSELL. J. Comp. Neurol. **35**:97, 1922.

The nerves which enter the lung from the anterior and posterior pulmonary plexuses become segregated within the organ near the hilum into three groups of plexuses, namely: (1) a bronchial group, (2) a vascular group, and (3) a plexus which is distributed to the pleura pulmonalis. The bronchial plexuses are two in number, an extrachondrial and a subchondrial; these fuse about the smaller bronchi. The extrachondrial plexus is composed of relatively large nerve trunks with many myelinated fibers and clusters of ganglion cells. The subchondrial plexus is composed of smaller nerve trunks; their unmyelinated fibers pass to the smooth muscles of the bronchi and their myelinated fibers to sensory terminations in the epithelium and to smooth muscle bands.

The intrapulmonary ganglions are found chiefly in the extrachondrial plexus and are characteristically located at the division points of the bronchi and larger nerve trunks. Their cells are surrounded by pericellular baskets representing terminal arborizations of preganglionic fibers of the vagus. A type of sensory termination which appears to correspond with the muscle spindles of striated muscle is present in the smooth muscle bands of the bronchial muscle. These terminations are designated smooth muscle nerve spindles. They are terminations of myelinated fibers of large size.

Nerve plexuses are present about the blood vessels, a rich plexus extending over the branches of the pulmonary artery, including the arterioles. The nerve fibers terminate in relation to the smooth muscle cells of the tunica media of these vessels. Sensory nerve endings are also present in the walls of the pulmonary artery near the hilum of the lung. The pulmonary veins and lymphatics have but few nerve fibers, but the relation appears to be the same as in the arteries.

The pleura pulmonalis receives nerve trunks from the periarterial plexus. The fibers, chiefly myelinated, pass to the margins of the pulmonary lobes. They terminate as sensory endings near the lobar margins, principally on the dorsal and interlobar margins of the lobes.

HERRICK, Chicago.

THE SIGNIFICANCE AND MANAGEMENT OF HYPOCHONDRIACAL TRENDS IN CHILDREN. ESTHER LORING RICHARDS, Ment. Hyg. **7**:43 (Jan.) 1923.

This is an interesting study and interpretation of hypochondriacal trends in twenty-two children treated at the Phipps Psychiatric Clinic. In age they averaged about 9.5 years, the youngest being 3 and the oldest 15. Their somatic complaints had existed from three weeks in recent cases to a period of nine years in chronic conditions. The symptomatic content faithfully reproduced the vocabulary of adult "invalids," but this in itself is scarcely astonishing nor conclusive since even in the presence of definite organic

disease, patients are often either restricted and vague or over-elaborative in their descriptive efforts. The actual somatic facts were few and relatively unimportant and in twelve instances the general health was normal. Under "reactional pattern" and "situational data" are described, respectively, the particular neurotic traits in adult relatives which the children appeared to mimic and the family setting, personal and environmental, which presumably favored the development of the neurosis. This is perhaps another way of stating one of the long established psychologic attributes of childhood; namely, imitativeness. In the cases cited, the genesis of the symptom complex is usually fairly obvious; occasionally the connection between it and the reaction pattern and situational data is somewhat obscure. The adjustment program is replete with common sense therapeutic recommendations which bore fruit in the recovery of eighteen of the twenty-two children.

Any neuropsychiatrist who has had experience in dispensary work will be able to substantiate the author's findings and agree that her methods are applicable to certain groups of patients. It will not detract from the value of the article to add, that the mechanism described is not by any means all-inclusive and that there are equally large groups consisting of children whose "hypochondriasis" is a definite response to serious organic ailments. Their readjustment is accomplished by close attention to the real physical needs.

STRECKER, Philadelphia.

EPILEPSY AND GUNSHOT WOUNDS OF THE HEAD. W. ALDREN TURNER, J. Neurol. & Psychopath. **3**:309 (Feb.) 1923.

There are here tabulated 38 cases of generalized, and 10 cases of focal epilepsy associated with gunshot wounds of the head. The patients were officers applying for compensation and were seen only once. They can be divided into two groups according as there was or was not a penetrating wound of the skull. In the former the seizures were both focal and generalized; in the latter they were of general character. All types of general seizures were noted—major, minor and "psychic." They were associated with lesions in all regions of the cerebrum: 10 of the frontal, 16 of the parietal, 9 of the occipital and 3 of the temporal lobe. It seemed probable that parietal injuries were more liable to result in epilepsy. No correlation was detected between the extent of bony injury nor the severity of the original cerebral trauma and the frequency of epilepsy.

An interesting feature is the observation that focal seizures developed early after the trauma and tended to disappear; they rarely passed over into the generalized form. They were frequently accompanied by paralytic and other phenomena of focal damage. The generalized seizures on the other hand, usually appear late—25 in the first year and 11 in from one to two and a half years in the cases in which the date of the first fit was known—after the injury, are relatively frequent and run a chronic course.

Turner is inclined to believe that traumatic epilepsy after gunshot wounds of the head is more frequent than the figures of Sargent and Holmes (5 and 6 per cent.) indicate and suggests that the proportion would be higher if a longer interval had elapsed before the investigation. He concludes that the epilepsy is not due to the local injury alone, but is inclined to ascribe much importance to neuropathic heredity. The cases reported were not investigated from this point of view; but of 9 cases of epilepsy with head injury seen in

civil practice, 4 gave a family history of epilepsy or mental defect. This factor is offered as an explanation for the unfavorable outcome in the series reported.

SINGER, Chicago.

STUDIES OF THE BRAIN STEM. VI. AN EXPERIMENTAL STUDY OF THE CORPUS STRIATUM OF THE PIGEON AS RELATED TO VARIOUS INSTINCTIVE TYPES OF BEHAVIOR. FRED T. ROGERS, J. Comp. Neurol. **35**:21, 1922.

Of the numerous experimental studies which have been directed toward functional localization in the forebrains of birds, most have been sadly deficient in adequate anatomic control. To avoid this defect Rogers studied microscopic preparations of the pigeon's brain before performing the extirpations described and at necropsy he supplemented the gross examination with study of serial microscopic sections in nearly every case. These experiments were preceded by prolonged study of the behavior of normal pigeons and by observations on the individual peculiarities of the specimens chosen for operation. Usually a complete mating cycle and rearing of young preceded the operations, but in some cases young squabs were operated on. The birds were kept alive after operation for several months, in a number of cases for two years or more with observation of breeding behavior.

The cerebral cortex of birds is very poorly differentiated, but the corpus striatum complex here attains maximum size and complexity of organization among vertebrates. These animals are, accordingly, favorable subjects for the analysis of the subcortical functions of the forebrain. In a series of pigeons decerebration operations were performed, from superficial lesions involving only cortical structures to practically complete removal of the cerebral hemispheres. The results are striking and very instructive.

The poorly differentiated cortical layer is thin except in the anterior part of the hemisphere and its cells are not arranged in layers as in mammals. The corpus striatum as described by Edinger includes a deep core-nucleus, the mesostriatum (probably part of the most primitive component of the striatum termed paleostriatum by recent writers), superficial to which are several large areas (ektostriatum, hyperstriatum, epistriatum, etc.), in more or less concentric arrangement.

Removal of one complete cerebral hemisphere produces no obvious permanent defects of behavior save for a certain degree of visual disturbance on the opposite side. Destruction of both frontal areas causes no observed difference in behavior. Loss of nearly all of the cortex without serious damage of the underlying striatum is followed by no disturbance of the usual laboratory behavior, including complete mating cycle and rearing of young. These reactions are largely instinctive. Whether under more natural conditions the individually acquired components of behavior would have been affected was not determined.

Destruction of the more superficial parts of the striatum complex (hyperstriatum) is followed by a long period of helplessness, but the bird may regain ability to feed itself. It does not go through the mating or nesting cycles of behavior. On the other hand, birds with hyperstriatal area intact but without the cortex can carry out a simple association or learning process involving the correlation of several different physiologic reactions to form the customary behavior cycle. In that bird in which there was the greatest loss of cerebral

tissue with subsequent regaining of ability to feed itself the ektostriatum, epistriatum and mesostriatum of one hemisphere were present, reduced in size. Injury to the mesostriatum involves serious disturbance of the fundamental reflexes.

HERRICK, Chicago.

FOLLOW-UP WORK IN MENTAL AND SURGICAL CASES. EARL D. BOND, Am. J. Psychiat. **2**:445 (Jan.) 1923.

The results of treatment in 377 consecutive mental patients after a lapse of five years are analyzed: 94 or 25 per cent. recovered; 68 or 18 per cent. were improved; 114 or 30 per cent. were unimproved; and 101 or 27 per cent. died. The manic-depressive group naturally yielded the greatest number of recoveries, sixty-one of a total of 160; the death rate was also high, 24 per cent.; thus, the acute and critical course of the affective psychoses is statistically emphasized. Dementia praecox is a chronic condition and of eighty patients only two had recovered and eight improved. Two elements of this work deserve particular mention. The first concerns the rigorous care that was exercised in passing judgment on the final status of a patient. Whenever a case presented even minor debatable points, it was excluded from the recovered list, so that the statistics may be used for basic comparisons. In the second place, there is included, a statistical contrast with the end results of surgery. In 314 surgical patients 30 per cent. recovered and 1 per cent. improved. There were 20 per cent. fewer unimproved, and 11 per cent. fewer deaths. In view of the facts that (1) five year surveys are not available in surgery and usually merely the direct operative outcome is taken into account; (2) that pathologic phenomena, either organic or functional, which arise subsequent to the operation, are generally not permitted to negative the effect statistically unless they are definitely related to the original disease either as complications or sequels, and finally, (3) that simple and local conditions such as a finger abscess or inguinal hernia or "clean" appendix which tend to increase surgical recoveries, scarcely have an analogue in psychiatry—it is easy to answer the author's query, "Is there any reason why a psychiatrist getting 25 per cent. recoveries, after illnesses which have drawn into action all the forces of the person, should not feel his work compares well with that of the surgeon who is getting 55 per cent. from general and partial illnesses?" with a decided negative.

STRECKER, Philadelphia.

CAUSALGIA AND ALLIED PAINFUL CONDITIONS DUE TO LESIONS OF PERIPHERAL NERVES. H. S. CARTER, J. Neurol. & Psychopath. **3**:1 (May) 1922.

This is a report of twenty-four (twenty-three gunshot wounds) cases of causalgia. The war cases occurred among 3,000 cases of peripheral nerve injuries, i. e., one in 130. Major causalgia occurred once in 750 cases. The author considers the diagnosis, differential diagnosis, pathogenesis, pathology, bacteriology, treatment (medical and surgical), and prognosis in detail. He tabulates the results of the study of the twenty-four cases, and gives photographs and microphotographs of the changes described. Carter concludes that the causal lesion in causalgia and allied conditions is one of intraneural and perineural sclerosis and that the irritation of the fibers set up at the site of the injury to the nerve results in perverted afferent impulses being sent to the cord and possibly to the subcortical and cortical centers, and thence efferent responses of vasodilator, secretory and trophic natures are reflected

to the peripheral distribution of the nerve where the reaction is interpreted as pain. He doubts that a special group of organisms causes this type of inflammatory reaction in nerves. The most that can be said is that there is some peculiarity in the trauma of the nerve in these few cases which causes disturbance of those cells in the brain whose function it is to interpret pain. By resection of the irritated and sclerosed portion of the nerve, the source of the perverted impulses is removed, the pain vanishes and regeneration remakes the path for normal impulses.

POTTER, Mercer, Pa.

ON CERTAIN FEATURES OF THE LATEROSENSORY CANALS OF THE PLAGIOSTOMI. EDWARD PHELPS ALLIS, JR., J. Comp. Neurol. **35**: 1, 1922.

The conflicting statements in the literature regarding the embryologic development and homologies of the lateral line nerves of fishes are reviewed. It is concluded that the laterosensory organs in *Plagiostomi* and *Teleostomi* differ radically, not only in the manner in which they become enclosed in their canals, but also in their relations to the dorsolateral placodes and in the manner of development of the nerves that supply them. The author does not consider the further possibility that among the *Plagiostomi* themselves there may be differences in mode of development and that these differences as described in various species may rest on diversities in actual fact and not on faulty observation. The recent observations of Bartelmez and others on the development of cranial ganglions in mammals suggest that this may well be the case and the entire question needs further comparative study.

Allis believes that the development of the lateral line nerves and ganglions suggests that these must be regarded as intrinsic to several primary segments of the head—trigeminal, facial, glossopharyngeal and vagal. He seems also to persist in an opinion formerly expressed, that terminal buds of fishes (cutaneous taste buds) and lateral line organs may be regarded as homologous structures or that their innervation in different species may be by homologous nerves. This latter view is quite at variance with the most careful embryologic and anatomic studies of recent years.

HERRICK, Chicago.

OTITIC ABSCESS OF THE CEREBELLUM. C. F. YERGER, J. A. M. A. **80**:244 (Jan. 27) 1923.

The case reported is a metastatic abscess of the cerebellum secondary to suppurative otitic disease in a man, aged 43 years. The present complaint began two weeks previous to his admission to the hospital with headache, which was almost continuous and involved the frontal and occipital regions and was associated with earache. He had had a chronic suppurative otitis media, involving the right ear, for the preceding eighteen years. Shortly after the onset of the headache he began to have attacks of vertigo, which were associated with falling to the right, but unaccompanied by nausea or vomiting.

Turbid fluid under pressure was found on lumbar puncture; there was increased globulin in the fluid and the cell count was 3,110 per cubic millimeter, with 96 per cent. polymorphonuclear cells.

An exploration of the right mastoid region revealed arrested development of pneumatization of the mastoid and the author calls attention to the fact that these cases are especially predisposed to the development of suppurative intracranial complications.

On operation an abscess was located in the right lobe of the cerebellum and 15 c.c. of thick, greenish yellow pus was evacuated. The patient died the following day.

NIXON, San Francisco.

THE PHENOMENON OF ABREACTION. R. G. GORDON, J. Neurol. & Psychopath. **3**:322 (Feb. 1923.

Conceding the therapeutic value of "abreaction" in the neuroses, Gordon here attempts to discover some basis for an intelligible statement of the nature and effects of the phenomenon. He points out the impossibility, in anatomic and physiologic terms, of an idea or a feeling being retained as such; and that a stimulus can only activate an engram which includes vegetative as well as central nervous system neurones. He discusses the six criteria involved in memory formulated by Lloyd Morgan—"register, registration, retention, revival, recognition or renewal with a sense of 'againness,' and reference in time and space." The first three can be observed in non-living matter; the last three require life. Recognition and reference to time and space are essentially conscious and involve the cerebral cortex.

Repression, suppression (Rivers) or forgetting is referred to a possible endocrine action on synapses, the glands being in some way activated by the emotion belonging to the experience repressed. When, in analysis through associations, a repressed memory is revived, recognized and referred, there is not only activation of the original engram, but also of vegetative activities resulting in "a feeling attached to the recognition" which is independent of the original engram and will account for the emotion experienced in the abreaction. The value of this 'psychocatharsis' depends on the recognition and reference to time and space, which imply cortical control and fusion with the personality, and is not due to a "freeing of bottled-up emotion."

SINGER, Chicago.

ANATOMICAL STUDIES ON THE MOTOR CORTEX OF MACACUS RHESUS. JUAN C. NAÑAGAS, J. Comp. Neurol. **35**:67, 1922.

In this anatomic examination of the motor cortex of the Philippine macaque monkey, the aim is to correlate structural localization with the results of previous experimental work on this species. After a preliminary survey of the gross relations of the Rolandic region, the microscopic structure of the precentral area is fully described.

The lamination pattern of the cortical cells differs in various parts of the physiologically excitable cortex. In this region there are three sizes of large cells of the Betz type in Brodmann's fifth layer. The largest are limited to a narrow zone in the anterior lip of the central fissure. The area of medium cells covers a wide field and the small cells are still more widely distributed. The area controlling the leg has the largest Betz cells; that controlling the arm has only medium and small cells. The fourth (granular) layer is absent in the area of large Betz cells and in most of the area of medium cells it is absent or reduced. The density of the pyramidal cells of the third layer is inversely proportional to the size of the underlying Betz cells and directly proportional to the thickness of the fourth layer. In those territories where the largest Betz cells are found (leg area) the pyramidal cells are more dispersed, probably because of the enormous development of the dendrites of these Betz

cells. These observations suggest that there is a topographic pattern of arrangement of cells in various parts of the motor cortex in correlation with functional diversity, and this point the author hopes to be able to subject to further experimental control.

HERRICK, Chicago.

ORGANIZATION AND SCOPE OF A STATE BUREAU OF MENTAL HEALTH. WILLIAM C. SANDY, Ment. Hyg. **7**:119 (Jan.) 1923.

Among the more important plans for the future outlined by the Pennsylvania State Bureau of Mental Health are the following: "(1) complete state care of mental patients as a goal to be attained gradually over a series of years; (2) increase of institutional capacity in accordance with a state-wide plan; (3) the establishment of at least two psychopathic hospitals for early diagnosis by intensive methods, to be utilized also as research and teaching centers for hospital physicians and other neuropsychiatrists; (4) establishment of facilities for the proper care and custody of defective delinquents; e. g., in separate institutions or in divisions of already existing institutions; (5) serious consideration of colonization of mental defectives, a method successfully carried on in other states; (6) continued endeavor to raise the standard of care and treatment of mental patients; (7) stimulation of vigorous efforts to rehabilitate more patients by parole and later discharge, aided by means of more clinics and more social workers in the hospitals."

Each one of these projects is worth while and of far-reaching import. Some of the county institutions are still hopelessly archaic in every respect and practically none of them are properly equipped or adequately staffed. As a class they scarcely attain to modern custodial standards. In the state hospitals, congestion makes scientific care and treatment an increasingly difficult problem. The establishment of psychopathic hospitals would make it possible to utilize an immense amount of valuable material for both teaching and research and would give impetus to intensive therapeutic efforts. Every psychiatrist should endorse Dr. Sandy's plans and give him whole-hearted support.

STRECKER, Philadelphia.

PONTOBULBAR CRISES ASSOCIATED WITH SIALORRHEA IN SYPHILIS OF THE NERVOUS SYSTEM. ERNST P. BOAS and WALTER M. KRAUS, J. Neurol. & Psychopath. **3**:329 (Feb.) 1923.

The case here recorded is that of a man of 35 who had been infected with syphilis at the age of 18. He presented irregular, Argyll Robertson pupils but no other sign of tabes. The tendon jerks were all active; there was no change in any form of sensibility and no Romberg sign. The blood Wassermann had been ++++, but under treatment became negative. The spinal fluid gave a doubtful Wassermann reaction, contained 7 cells and positive globulin, and the colloidal gold test on two occasions gave the results: 0133100000 and 0111000000.

The principal symptoms were severe crises characterized by: (1) profuse secretion of thin and watery saliva; (2) gastric hypersecretion; (3) attacks of vomiting related to salivation; (4) tachycardia; (5) attacks of high blood pressure; (6) sensation of bad taste in the mouth; (7) pain in the suprasternal notch radiating downward and to the left; and (8) diminution of pharyngeal and absence of laryngeal reflexes. The authors incline to the view that these

symptoms are of central origin and are due to involvement of the dorsal, visceral vagus nucleus; the adjacent sensory vagus nucleus; the fasciculus solitarius; the nuclei salivatorii of Kohnstamm; and the glossopharyngeal and vagus nuclei. The article is well illustrated and a review of the literature is given.

SINGER, Chicago.

THE DILATOR PUPILLAE AND THE SYMPATHETIC. N. WILLIAM INGALLS, J. Comp. Neurol. **35**:163, 1923.

Attention is called to certain physiologic differences between the neuromotor apparatus of contraction and dilation of the pupil. The sphincter mechanism is more specialized in every way and is of far greater importance in vision than the dilator. The two muscles differ in mode and time of development and in adult histologic structure. The sphincter is a stronger muscle and it contracts more rapidly; indeed, the oculomotor mechanism alone seems quite equal to the task of regulating the pupil for purely visual purposes, for this function can be carried on after total paralysis of the dilator nerves.

The dilator of the iris, on the other hand, has a two-fold function—first, and probably more primitively, a visual function, and second what may be called the sensory reflex, that is, reactions ordinarily elicited by cutaneous stimulation. Dilation of the iris comes into play as part of a complex reaction which includes smooth muscle of cutaneous vessels, hairs or glands, and various visceral activities, all of these being frequently associated with emotional disturbance. "The actions of the sympathetic (*sen. str.*) on the eye are all calculated to render it more conspicuous and impressive to the beholder, the differences for the possessor are negligible."

HERRICK, Chicago.

THE CONSTITUTIONAL PSYCHOPATHIC INFERIOR—A PROBLEM IN DIAGNOSIS. ALICE E. JOHNSON, M. D., Am. J. Psychiat. **2**:467 (Jan.) 1923.

Johnson attempts a sharp differentiation between the constitutional psychopathic inferior and the psychoneurotic person on the basis of their reaction to fear. The former do not profit by the experience of pain; and the quality, which may be termed moral impressionability, stands at a very low level. Consequently, there is complete failure of adjustment. "The condition is seen in very young children and in all ages thereafter. These people come from every class of society; they may have a higher or lower grade of intellectual ability, may be organically defective or perfect specimens of physical development. In manner they are often pleasant and agreeable on first appearance. Except when frustrated in some desire they are good natured enough. But they are bad citizens and they do not improve. They forget the past and think hopefully or not at all of the future. In short, they are motivated almost entirely by the desire of the moment. Their inhibitions are weak and evanescent." On the other hand, psychoneurotics also fail to meet the ordinary demands of socialization, but for a different reason. They have too much apprehension and their symptoms and maladjustment spring from endless and hopeless efforts to produce an "illusion of safety." From the standpoint of prognosis the author feels that the constitutional psychopathic inferior cannot be cured because the defect is inborn and not acquired. Psychoneurotics may be readjusted.

STRECKER, Philadelphia.

OBSERVATIONS ON CREATINURIA AND GLYCOSURIA IN MYASTHENIA GRAVIS. B. W. Williams and S. C. Dyke, Quart. J. Med. **15**:269 (July) 1922.

In four cases of myasthenia gravis investigated, creatinuria was present. One case showed considerable amounts of creatin in the urine when the patient was on a creatin-creatinin free diet. Two patients were unable to deal with small amounts of creatin when given by mouth. In two cases the percentage of muscle creatin was estimated from biopsy specimens and was found to be low. In all four cases the glucose tolerance was lower than normal. Williams and Dyke conclude that the defective muscles are responsible for the altered carbohydrate tolerance in myasthenia gravis and that the creatinuria observed in these four cases was probably secondary to this defective glucose metabolism.

Potter, Mercer, Pa.

CASE OF CEREBRAL NEOPLASM SIMULATING DEMENTIA PARALYTICA, WITH OPERATION AND COMPLETE NEUROLOGIC AND MENTAL RECOVERY. Charles Rosenheck, J. A. M. A. **80**: 470 (Jan. 17) 1923.

The patient, whose case is reported in detail, presented the neurologic signs and psychiatric picture of an intracranial growth in a manner so vivid and clear cut that the diagnosis and localization were a matter of comparative ease. The mental changes simulated those of dementia paralytica quite faithfully. A large endothelioma of the right frontoparietal area was removed and at the time of the report, six months after the operation, the patient was apparently in excellent physical and mental condition.

Nixon, San Francisco.

ORIGIN AND DISTRIBUTION OF THE TRACTUS SOLITARIUS IN THE GUINEA-PIG. William F. Allen, J. Comp. Neurol. **35**:171, 1923.

This is part of an extensive experimental program directed toward the analysis of gustatory and general visceral conduction pathways in the mammalian brain. The roots of the seventh, ninth and tenth cranial nerves were torn out, each in a different animal, and in other cases the fasciculus solitarius was cut across in its intrabulbar course. The resulting degenerations were studied by the Marchi method. The result is a very exact determination of the central courses of each of the sensory systems of fibers which enter the fasciculus solitarius.

No facialis fibers descend in the fasciculus solitarius below the level of entrance of the first vagus rootlet, and no glossopharyngeal fibers descend below the level of entrance of the third sensory vagus rootlet. The nucleus accompanies the tract for its entire length. That part of it lying rostrally of the third vagal rootlet receives practically all of the gustatory fibers. The greatly expanded spinal end is probably devoted to general visceral functions. A second paper on the degenerations resulting from lesions of the nucleus of the fasciculus solitarius is in the press.

Herrick, Chicago.

SPIROCHETOSIS OF THE CEREBROSPINAL FLUID. D. O. Riddel and R. M. Stewart, J. Neurol. & Psychopath. **3**:345 (Feb.) 1923.

The spinal fluids of 23 cases of general paralysis—7 acute, 16 subacute or chronic—were examined with dark-field illumination immediately after with-

drawal and also in smears from the deposit obtained by centrifugation at low speed. The latter were stained with the Giemsa or the Fontana-Tribondeau silver method. Successful results were also obtained by impregnating the deposit by Jahnel's method and embedding in paraffin.

The results were negative except in one case, the fluid from a lad of 20 with juvenile general paralysis. Spirochetes were discovered on six consecutive punctures during a period of forty-one days. Thereafter they disappeared and could not be found in twelve more punctures. Fluid obtained by cisternal puncture contained more organisms than that by lumbar puncture. Attempts at cultivation were unsuccessful.

SINGER, Chicago.

THE MENTAL HEALTH OF 463 CHILDREN FROM DEMENTIA-PRAECOX STOCK. MYRTELLE M. CANAVAN, M.D., and ROSAMOND CLARK, Ment. Hyg. **7**:137 (Jan.) 1923.

Canavan and Clark investigated the mental health of the offspring of dementia praecox stock. Of 381 children, the product of 186 matings, 86 deviated from the normal, either mentally, physically, or socially. The deviators consisted of: 5 dementia-praecox patients; 4 feebleminded; 12 backward; 12 nervous; 17 physically diseased, and 36 with conduct disorders. The mother had dementia praecox in 74 and the father in 12 cases. Two hundred and fifty schizophrenic mothers and 45 fathers mated with presumably sound stock and produced 295 normal children. The authors recognize a potential wide margin of error arising from the fact that 68 per cent. of the deviated group and 79 per cent. of the normals were less than 16 years old. It is not improbable that the further statistical reviews which are contemplated in 1925 and 1930 will increase the number of deviates.

STRECKER, Philadelphia.

INTRASPINAL THERAPY IN NEUROSYPHILIS. J. A. FORDYCE, Am. J. Syphilis **6**:198 (April) 1922.

Fordyce asserts that "intraspinal treatment cures certain types of neurosyphilis in which intravenous treatment alone or when combined with spinal drainage has failed," and bases his assertion on four years of observation of the method. Fordyce does not drain the fluid preliminary to the introduction of arsphenaminized serum, but effects its introduction under pressure; he has found that the clinical and serologic results are better without preliminary drainage.

VONDERAHE, Philadelphia.

A NOTE ON THE EXCITABLE AREAS OF THE CEREBRAL HEMISPHERES OF THE PIGEON. FRED T. ROGERS, J. Comp. Neurol. **35**:61, 1922.

This brief article reports experiments on the motor responses derived from electric excitation of the cerebral hemisphere of the pigeon. There is no excitable cortex related to the control of skeletal muscle analogous with that of mammals. Two definite cortical responses involving the sympathetic nervous system can be secured: (1) constriction of the pupil of the contralateral eye from an area at the dorso-medial border about midway between frontal and occipital poles of the hemispheres. (The same effect can be obtained, but less certainly, from the occipital cortex.) (2) Depression of the feathers of

the facial region from a frontal area close to the olfactory bulb. Besides these strictly cortical motor areas, motor responses to deeper stimulation of the underlying corpus striatum are described: (1) movements of the beak from the ektostriatum; (2) bilateral winking of the eyelids, also from the ektostriatum.

HERRICK, Chicago.

VENTRICULOSCOPY AND INTRAVENTRICULAR PHOTOGRAPHY IN INTERNAL HYDROCEPHALUS. TEMPLE FAY and F. C. GRANT, J. A. M. A. **80**:461 (Feb. 17) 1923.

The authors did ventriculoscopy and intraventricular photography in a case of hydrocephalus and found that these procedures were followed by little or no reaction when properly conducted.

Direct inspection of the ventricles may assist in determining the location and the extent of growth of subcortical lesions causing deformities of the ventricles and will permit callosal puncture under direct observation.

NIXON, San Francisco.

CATATONIC DEMENTIA PRAECOX; PHYSIOTHERAPEUTICS, AND RESULTS OBTAINED IN A SERIES OF TWENTY CASES. DANIEL C. MAIN, Am. J. Psychiat. **2**:473 (Jan.) 1923.

Dr. Main has attacked the problems of profound inertia, mental deterioration, and untidiness in a group of twenty cases of catatonic dementia praecox. He has not tried to give the patient an occupation by which he can earn a living outside of the hospital, but by which he can adjust to the hospital. Brief histories are given of twenty patients in the second and third decades, illustrating change of attitude through interest in work and incidentally recording several recoveries. Dr. Main's remark in his discussion is illuminating: "I think controls can be found on the 'back' wards of any of our hospitals."

BOND, Philadelphia.

Society Transactions

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, April 19, 1923

H. I. DAVIS, M.D., *President, in the Chair*

ACHONDROPLASIA IN MOTHER AND CHILD (Illustrated with lantern slides). DR. PETER BASSOE.

A little woman. aged 31, was admitted to my service in the Presbyterian Hospital on account of weakness of the right arm and leg which had been coming on for over a year. It was apparent at once that she was a typical short-limbed dwarf. Her total height was 130 cm., (51⅛ inches); height at the shoulders 100 cm., at the iliac crest 60 cm.; weight 125 pounds (57 kg.). Her head was relatively large, its horizontal circumference being 56.6 cm.; the forehead was prominent, the insertion of the nose rather deep; the head was distinctly brachycephalic. The length of the upper extremities from the acromion to the tip of the middle finger was 53 cm.; of the lower extremity from the anterior superior spine of the ilium to the inner malleolus, was 56 cm. The second and third toes were partly webbed. The length of the fingers was as follows: First 5 cm.; second 7.3 cm.; third 7.8 cm.; fourth 7 cm.; fifth 5.5 cm. The appearance was that of a trident hand. The patient was well nourished. The breasts and genitalia were well developed and the hairy development and distribution were normal. Roentgenologic examination by Dr. Cassie B. Rose, showed the base of the skull to be very short, measuring 8.5 cm., while the vault measured 14.8 cm. in height, and 21.5 cm. in the greatest anteroposterior diameter. The front region was protruding and rounded; the suture lines were still open and the vascular markings prominent; the sella was of normal size, with well defined margins; because of the short skull base, the nasal region appeared to be pushed inward but there was no unusual alinement of the teeth. The basal angle was acute, representing a basal kyphosis which probably accounted for the apparent retraction of the face and nasal region. Practically the entire skeleton was examined and, aside from the shortness of the long bones, the most striking features were: the shortness of the neck of the femur, and the high position of the head of the fibula in relation to the knee joint.

The patient was married. The first child was still-born but the second, aged 4½, was delivered by cesarean section. This child is a girl, 86.5 cm. tall, whose head is of the same shape as that of the mother. Her arms are short, reaching only to the lower end of the trochanter. The lower extremities are not strikingly shortened, but the head of the fibula is also high. The circumference of the head is 51 cm. In the roentgenogram the base of the skull measures 7 cm. There seems to be an unusual amount of ossification about the base, particularly beneath the sella. The sphenoidal sinus cannot be made out. There is an irregular excavation under the anterior clinoid process, similar in appearance to that described by Timme as characteristic of mongolian idiocy. The suture lines are open and vascular markings are

prominent. This child is remarkably agile and hypotonic. She can separate the legs and slide down on the floor with the legs at a right angle to the trunk and touching the floor along their entire inner borders.

The nature of the hemiparesis of the mother was not determined. We were in doubt as to whether it was caused by the slight chronic hydrocephalus, which has been shown by Dandy to be the cause of the enlargement of the head in this condition, or whether it was hysterical.

The cause of achrondoplasia remains unknown as we cannot accept the recent hypothesis of Murk Janson that it is due to abnormalities of the amnion. The presence of the skeletal deformities early in fetal life makes it difficult to attribute the condition to endocrine disorders. However, one is struck by the fact that these dwarfs are just the opposite of eunuchs or eunuchoid individuals; the dwarfs have large heads, long trunks, short extremities and, usually, excessively developed genitals and muscular system; the eunuchs have small heads, short trunks, long extremities, defective genitals and flabby muscles. These contrasts do not suffice to stamp the condition of achrondroplasia as due to hypergenitalism, but the latter apparently is a feature of it.

TWO SPECIMENS OF OCCIPITAL LOBE LESIONS. Dr. John Favill.

Recently two cases of occipital lobe lesions in the same ward of the County Hospital came to necropsy during the same week, and it was thought that a brief review of their histories, together with the gross specimens, might be of interest.

Case 1.—A negro, aged 37 years, entered the hospital, Feb. 22, 1923, complaining of headache, vomiting, dizziness and "falling spells," which had gradually developed during the preceding three weeks. He remembered that six months ago his left leg once gave way. The headache was "like a vise" at times and the vomiting would come without warning. It was noted that his pupils did not react to light, but he refused to permit further examination and went home. He entered again March 7, being in a rather clouded mental condition with frequent spells of real coma. He complained of ringing in the left ear of two months' duration. The previous complaints were accentuated.

Examination revealed: deviation and falling to the left on attempting to walk; ataxia of the left arm; weakness of the left arm and leg, with increased tendon reflexes; absent cremasteric and abdominal reflexes and positive Babinski sign on the left. Sensation could not be satisfactorily tested. The pupils were immobile to light and there was some weakness of the left seventh nerve. Speech was normal, but memory for recent events was bad. A preliminary diagnosis of cerebrospinal syphilis was made, but the Wassermann reaction with blood and spinal fluid and the colloidal gold test proved to be negative. The cell count was 6 and tests for blood and globulin were negative. The diagnosis was changed to left cerebellopontile angle tumor. A few days later, left homonymous hemianopia was noted. Optic neuritis developed and was more severe on the right. Attacks of stupor and coma came more frequently and on March 20, he went into coma from which he did not rally. Two days later his eyes were directed up and slightly outward; the head was turned rigidly to the left and showed an interesting dissociation of the usual occulocephalogyric mechanism. There was no apparent facial paralysis. A final diagnosis of tumor of the right hemisphere was then made, further localization being difficult. He died March 24.

The necropsy showed tension in both occipital lobes. The spinal fluid was clear. On cutting the right parieto-occipital lobe, yellow fluid escaped from a cavity. The specimen shows a soft gelatinous finger-like projection in this cavity, and behind the projection is firm tumor tissue. The right lateral ventricle is completely collapsed and the left dilated. The anatomic diagnosis is glioma with cystic and colloid degeneration.

Case 2.—A Pole, aged 34 years, entered the hospital Dec. 27, 1922, complaining of headache and right-sided weakness which began three weeks previously. Two months before this, he had had an abdominal operation, probably for appendical abscess, judging from the scars of McBurney and right lumbar incisions. The right leg was weak and spastic but without definite tendon reflex increase or positive Babinski sign. Abdominal and cremasteric reflexes were absent. General sensibility and cranial nerves were normal, the pupils being regular, equal and reactive to light. The heart, lungs and abdomen gave negative findings. The spinal fluid cell count was 200 with positive globulin and negative Wassermann reactions, but the blood Wassermann reaction was positive. A preliminary diagnosis of cerebrospinal syphilis was made and antisyphilitic treatment was begun. A week after entrance, right homonymous hemianopia was observed and the diagnosis was then elaborated as hemorrhage or thrombosis involving the posterior portion of the left internal capsule. He gradually improved under continued antisyphilitic treatment during January, but began to fail in February, going into occasional states of stupor. He developed a cough in March with sanguinopurulent sputum and signs of cavity formation in the right lung between the scapula and spinal column. Roentgen-ray examination revealed a lung abscess on the right with probable tuberculous bronchopneumonia. The sputum showed streptococci, bacilli and diplococci, but no tubercle bacilli. He died March 23, 1923.

The necropsy revealed extensive gangrene of the right lung. There was brown fluid in the right sphenoid cells. Hemolytic streptococci were cultured from the pleural fluid, spinal fluid and the infected sphenoid. The specimen shows a piece of adherent dura over the left parieto-occipital region of the brain. Under this, and beneath the cortex, is a definite abscess cavity about 2 cm. in diameter. Several other smaller cavities can be found in the neighborhood.

DISCUSSION

Dr. Peter Bassoe: I have looked up records of six cases of occipital lobe tumor that came to necropsy. Each one presented some difficulty or other in diagnosis. The first patient was a young man, a heavy drinker, who complained of headache, occasional dizziness, and sometimes a symptom described by Dr. Favill; namely, a feeling of going right down. The disks were normal. I saw him at my office and sent him to the hospital thinking that the case was either alcoholic dementia or general paralysis. When I reached the hospital the next morning, the intern had found that he had hemianopia. He died within two days. A glioma of considerable size was found in the occipital lobe.

Another case was that of a nurse, aged 60, who had worked very hard. She had been in the hospital for some time, was considered a case of neurasthenia and had been allowed to leave. She came in again and at that time had distinct delusions. I thought it a case of presenile paranoid state. There were no definite physical signs. Just when she was to be sent to a sanitarium,

she had a sort of stroke and developed a partial hemiplegia. She was so ill mentally that it was impossible to test the optic fields. Necropsy disclosed a glioma in the occipital lobe.

Another case was in a man who worked for a railroad company, had had a minor injury and complained of dizziness. The fields were normal on rough test and the disks were normal. We thought it a case of traumatic neurosis. Some months later, necropsy disclosed a tumor of the occipital lobe. I understand that hemianopia had developed before death. I saw him only in the early stage and it was almost impossible to determine whether he had an organic brain lesion.

Another case was in a man, aged 60, who had heart disease and bronchopneumonia. He was very weak and complained a great deal of headache. He grdually developed a left hemiplegia; hemianesthesia and hemianopia were also present. At no time was there any change in the disks. While I suspected abscess, I was rather inclined to think it a case of thrombosis. At necropsy we found a glioma in the occipital lobe with extensive softening.

An elderly man had definite signs of an occipital lobe lesion. There were hemianopia and hemiplegia. The spinal fluid gave a positive Wassermann reaction, but the colloidal gold reaction was far to the right. His brain was examined postmortem and metastatic carcinoma was found.

The sixth patient was a young man who complained of headache, with only general tumor symptoms and a great deal of occipital pain. Subtemporal decompression was immediately followed by hemianesthesia and hemianopia. Necropsy revealed a large endothelioma of the dura that had started at the torcula Herophili and filled the longitudinal sinus. Undoubtedly the shifting of the brain at the decompression caused the hemianopia to appear so suddenly.

My experience has been that occipital lobe tumors, which ought to be easy of diagnosis, often give a great deal of difficulty.

DR. YUDELSON: Were sensory changes found in the second case which would suggest a diagnosis of an internal capsular lesion?

DR. L. J. POLLOCK: In Case 2, although there was hemiparesis of three weeks duration, the history given me was that the paralysis had developed slowly within a period of less than two days. On examination he had a complete right hemiplegia, hyperesthesia and right homonymous hemianopia. In the absence of other findings it looked like a case of cerebral thrombosis involving the posterior limb of the internal capsule. Pulmonary changes were not detected until three or four days before death. Looking back one can see how this mistake could have been avoided. The hemiplegia was incomplete and the sensory changes were very slight; hemianopia was complete, such a dissociation of these three symptoms should have led to the inference that the lesion was not capsular, but further back, so that some motor and sensory fibers could escape.

DR. H. D. SINGER: I am impressed with the striking uniformity in the occurrence of mental symptoms. Both cases of Dr. Favill and most of those mentioned by Dr. Bassoe presented stupor and lack of cooperation. This observation in occipital lobe lesions is of interest and I wish Dr. Favill would give any further details of the mental state he may possess.

DR. FAVILL (closing): I am sorry that I cannot give any important details of the mental condition. There was poor cooperation, frequent stupor and little material on which to base any conclusion. It seems to me that the lesson to be learned from these cases is the importance of testing for hemianopia,

even under conditions in which only the roughest estimate can be made. This was true in the first case and the finding was of more help than anything else in giving an idea as to localization.

NEUROPSYCHIATRIC ANALYSIS OF 2,500 EX-SERVICE MEN WITH SPECIAL STUDY OF THE RELATION OF THE THYROID TO PSYCHONEUROSES. Dr. Ralph P. Truitt.

Eleven thousand ex-service men were discharged from the army in the Chicago district, which includes the States of Wisconsin, Michigan and Illinois, because of neuropsychiatric disability. Of this number 6,000 had been examined before December, 1922. Two thousand five hundred cases were examined (many of them reexamined) during a period of little more than a year while I was in charge of the neuropsychiatric examining and out-patient treatment departments of the U. S. Veteran's Bureau in Chicago. This group of cases which, in many instances, had the advantages of hospital and laboratory study has been analyzed diagnostically. The study revealed the following diagnoses:

Psychoneuroses	1,238
Constitutional Psychopathic Personality	237
Dementia Praecox	190
Neurosyphilis	133
Nerve Injuries	102
Mental Deficiency	98
Dysthyroidism	83
Cerebral Injuries	45
Epilepsies	43
Manic-Depressive Psychoses	22
Post-Encephalitic Conditions	20
Facial Paralyses	19
Post-Meningitic (Epidemic) Conditions	18
Sciatic Nerve Involvement	16
Multiple Sclerosis	12
Migraine	8
Raynaud's Disease	7
Spinal Muscular Atrophy	5
Pituitary Disorders	4
Poliomyelitis	4
Cerebral Tumor, Syringomyelia, Trifacial Neuralgia, Optic Nerve Blindness, Nerve Deafness and Spinal Sclerosis, 3 each	18
Muscular Dystrophies, Myatonia Congenita, Myelitis, Multiple Neuritis, Arteriosclerosis and Cranial Nerve Injuries, 2 each	12
No Neuropsychiatric Disorder	131
Undiagnosed	35
	2,500

Over one-half (701) of the psychoneurosis group were anxiety neuroses or anxiety states, about one-third (391) were neurasthenia, 128 were hysteria, and 18 were cases of psychasthenia. The great number of psychopaths may be accounted for by the fact that Chicago is a gathering point for "hobos" and other examples of the country's floating population. Many psychotic cases were already confined in institutions, which accounts for the small number found at this date. The syphilis group embraces: cerebrospinal syphilis, 102 cases; general paralysis, 17 cases; and tabes, 14 cases. The nerve injury group was about equally divided between the right and left sides of the body, but

there was a 3:1 ratio between upper and lower halves of the body. Enlargement of the thyroid gland was noted in 346 cases. Metabolic ratings were made in 86 cases and there were 19 thyroidectomies.

Analysis of the enlarged thyroid cases revealed:

Anxiety Neuroses	164
Neurasthenia	82
Hyperthyroidism	26
Exopthalmic Goiter	14
Hysteria	13
Constitutional Psychopathic States	12
Dementia Praecox	7
Cerebrospinal Syphilis	6
Goiter	6
Miscellaneous	16
Total	346

Subjective causes for the condition found on examination, as given by sixty claimants in the cases on which metabolic ratings were made were: High explosives, fright, etc., 18; exposure and overwork, 9; gassing, 8; wounded and fright, 7; influenza, 6; gassed and wounded, 5; influenza-pneumonia, 4; and vaccine injections, 3.

The chief subjective and objective symptoms of the cases with metabolic rating, in order of frequency, were: Tremor, anxiety, tension, fatigue, rapid cardiac action, cardiac distress, respiratory difficulties, sweating, increased deep reflexes, disturbed sleep, dizziness, cyanosis and flushing, gastro-intestinal, weakness, fainting, vague pains, ocular signs, headaches, frequent urination, loss of weight, twitchings and lack of mental concentration. A study of the metabolic ratings (made by National Pathological Laboratory with the Jones apparatus) is of interest and is shown in the accompanying table:

BASAL METABOLISM RATES

	Increased					Diminished			
	90	61-80	41-60	21-40	11-20	1-10	Normal	1-10	11-20
Exophthalmic Goiter	1	2	1	—	—	—	—	—	—
Hyperthyroidism	—	—	2	3	4	—	—	—	—
Anxiety Neuroses	—	—	—	3	10	17	2	3	—
Neurasthenia	—	—	—	—	—	7	4	12	4
Simple Goiter	—	—	—	—	—	—	—	1	1

Analysis of the present condition of the thyroidectomy cases revealed: Three worse; ten, no change; four, improved; and two recovered. One of these cases was cerebrospinal syphilis; one was considered to be hysteria; two were cases of psychopathic personality; four were neurasthenia; four had anxiety neuroses; three had hyperthyroidism; and four were cases of exophthalmic goiter. One of the exophthalmic goiter cases, following ligation of one thyroid artery, had a complete hemiplegia.

The similarity of symptoms in anxiety neuroses to those of hyperthyroidism and thyrotoxicosis is sufficient to call for further study, especially as increased metabolic rates may be found in anxiety conditions. The possible relation of emotional states and the sympathetic nervous system to the thyroid gland have been mentioned by various authors. The anxiety neuroses may be associated with hyperthyroid activity, but neurasthenia, the opposite of anxiety as a "nervous" condition, would appear to be more commonly associated with hypothyroid activity. The relation of anxiety conditions to hyperthyroid states and enlargement of the thyroid is not definitely known. One lieutenant in

the air service was found to have exophthalmic goiter (metabolic rate + 85 to + 108) ten months after falling from the air in a burning airplane, while on his second flight. He was not injured and continued flying, but was anxious, fearful, tremulous, easily fatigued, and uneasy. Ten months later, he had cardiac distress, exophthalmos and moderate general enlargement of the thyroid gland. The right lobe and half of the left lobe were excised sixteen months after the accident with gradual improvement and fair recovery in one year. Three weeks before the accident he had passed an examination for oversea service and there had been no intercurrent illness. There were other similar cases, less definite and acute; such cases certainly need explanation.

In many of the cases studied, the onset was acute, in others more gradual; still others were considered to be of emotionally unstable constitution. Generally, the thyroid enlargement was noted several months, even a year or two, after the initial anxiety. There was a notable absence of negroes. The education of those studied was above the average and many gave no history of previous anxiety. The psychoneurotic and emotionally unstable cases in general showed fluctuating metabolic ratings, and reduction of the higher rates accompanied improvement in the mental condition. One man with a typical anxiety condition had a + 39 metabolic rating. A few of the cases had a definite history of anxiety hysteria at the onset, but now presented the usual neurasthenic picture. Some patients dated the onset of their condition after the armistice or on return home after discharge. Some were examples of "compensation (anxiety) neurosis."

The anxiety patients were, in many respects, similar to those with thyroid disease; they were alert, tense, unable to relax, easily excited, frightened, explosive and expectant. The neurasthenics, on the other hand, were rather slow and phlegmatic and made numerous vague complaints that were out of keeping with the actual findings; they were perpetually tired and weak, instead of showing exhaustion after an episode of excitement, as observed in the thyroid and anxiety cases.

Estimation of basal metabolism is a great aid in differentiating the psychoneurotic from the active thyroid disorders, but one rating is often not sufficient. The size of the thyroid means little. Distinct and repeated elevation of the metabolic rate is the best criterion for diagnosing hyperthyroid states proper. One cannot but feel that anxiety and emotional states have important causal relations to dysthyroidism of all grades; this phase of the question should be more thoroughly studied and not passed over by applying the terms "neurasthenia" and "nervousness."

DISCUSSION

DR. MARKWALD (by invitation): Our laboratory made the majority of the metabolic determinations in these cases, some of which were sent back for repeated examination. In the cases presenting the "anxiety" factor, one could not help noticing the lowering of the metabolic rate on the second, or later examination. As regards the pulse rate, I am thoroughly in accord with Dr. Sanford of the Mayo Clinic who believes that there is a definite relation between the pulse rate, pulse pressure and metabolic rate. We found that, barring other complications, there was usually a very definite ratio between the pulse and metabolic rate. The pulse rate at the time the determinations were made, was uniformly lower than was found by Dr. Truitt. This, of course, was due to the fact that the patients were in all cases kept as quiet as possible. But, despite this fact, second and subsequent determinations in these cases showed lower pulse and metabolic rates.

THE PATTERN OF WEAKNESS OF THE HAND IN ULNAR AND MEDIAN NERVE LESIONS. (Illustrated with lantern slides). Dr. Lewis J. Pollock.

(1) *Ulnar Nerve Lesions.*—Physiologic interruption cannot be differentiated from anatomic section by the strength of movements of the phalanges of the fingers. In recovering and partial lesions, relatively greater strength is observed in the phalanges, but may at times be an inaccurate guide to the severity of the lesion. Relatively greater strength in the first dorsal interosseous, or in the abductor of the little finger, is an accurate guide to the incompleteness of a lesion. Of course, any movement of the flexor carpi ulnaris or adductor of the thumb, which is not supplementary in character, determines an incomplete lesion. Of sixteen partial or recovering lesions, twelve showed motor phenomena indicative of the severity of the lesion. In nine severe cases not due to anatomic section, the motor phenomena were suggestive of partial lesion in five, but conclusive in none.

Of sixteen cases of recognized partial or recovering lesions, eleven showed incomplete sensory loss. Sensation was completely lost in all severe partial lesions. When sensory regeneration had occurred, motor recovery usually could likewise be demonstrated.

(2) *Median Nerve Lesions.*—Physiologic interruption cannot be differentiated from anatomic section by the strength of the movement of the phalanges of the fingers. Although in a considerable number of cases the movements of the index finger were stronger, this could not alone determine the character of the lesion. Return of function in the opponens pollicis would indicate a partial or recovering lesion, but because of supplementary motility this is very difficult to determine. I wish to call attention to a supplementary movement producing adduction of the thumb at right angles to the palm which I have not before noted. When the metacarpophalangeal joint of the thumb is partially ankylosed, so that no flexion or extension in the plane of the palm is possible, contraction of the extensor longus pollicis and the extensor ossis metacarpi pollicis produce abduction of the thumb as above described.

Sensory regeneration or incomplete sensory loss in the area supplied by the median nerve is almost constant in incomplete lesions and in otherwise physiologically complete lesions; i. e., sufficiently severe partial lesions to come to operation. In a large proportion of partial or recovering lesions sensory regeneration is present when motor phenomena give no indication of regeneration.

3. *Combined Ulnar and Median Lesions.*—When observed more than five months after injury, it would appear that anatomic section of both ulnar and median nerves produces complete paralysis of all the phalanges of the fingers and thumb, and severe lesions, not anatomic sections, show some movement in some of the phalanges of all of the fingers.

In incomplete lesions of either ulnar or median nerves, weak movements of the phalanges of the fingers, if interpreted alone, are sufficient guides as to whether one of these nerves is severed and as to which one is severed.

As in isolated lesions of the median nerve, so when combined with an ulnar nerve lesion, it was seen that in partial or recovering lesions, and in more than half of the severe lesions that were not anatomic sections, incomplete sensory loss in the median distribution was present.

Only a few of the cases of incomplete ulnar and median nerve lesions showed incomplete sensory loss in the ulnar distribution and in only one of

seventeen cases was this the case when phenomena of motor regeneration were present. Only one severe incomplete lesion showed incomplete sensory loss of the ulnar. In complete lesions of the ulnar and incomplete lesions of the median, when sensory regeneration was demonstrable, motor regeneration was likewise present.

It is emphasized that recovering or incomplete lesions of the median nerve may almost regularly be determined by sensory examination, whereas in ulnar lesions this rule does not apply, contrary to the generally accepted statement that signs of sensory regeneration are first to appear.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting April 27, 1923

CHARLES M. BYRNES, M.D., *President in the Chair*

A CASE OF JACKSONIAN CONVULSIONS PROBABLY FROM CORTICAL ANGIOMA. DR. A. E. TAFT.

This case will be reported in full in a paper published elsewhere.

A CASE OF EPIDEMIC ENCEPHALITIS EXHIBITING A CONDUCT DISORDER. DR. F. H. LEAVITT.

This case is from the out-patient department of the Philadelphia Orthopaedic Hospital and Infirmary for Nervous Diseases on the service of Dr. Charles W. Burr. The patient is a white boy aged 17, born in this country of Russian Jewish parents, whose family and past medical history are negative except that a sister, has a chronic spasmodic tic of the shoulder. The present illness began in April 1922, with a fever of from 101 to 102 and a "queer feeling in his head." He was confined to bed for five days, suffering from insomnia at night and somnolence during the day. There was no diplopia, muscular twitching, or incontinence at any time, but he was physically very weak. In two weeks he returned to school, but was unable to study because of continual drowsiness during the day with insomnia at night. Six weeks after the initial fever he began to exhibit attacks that were described by his mother as occurring at any time of day, in which he suddenly held his head; his face became immobile; his mouth opened and saliva dribbled from the corners; and he was unable to talk or give a reply to questions asked. He appeared as though unable to comprehend what was said to him and he had an overpowering desire to lie down wherever he might be. These "attacks," as his mother called them lasted sometimes for only a few minutes or for days, and eventually became fixed.

When he appeared in the clinic of the Orthopaedic Hospital in November, 1922, his gait and attitude were those of Parkinson's disease. He held his hands stiffly in front of him without tremor, and saliva dribbled from his mouth. All muscular movements were "waxily" performed and his attitude and mental state at times resembled the catatonia of dementia precox. His bodily configuration, distribution of hair and subcutaneous fat were of feminine type; all tendon reflexes were equally hyperactive but not spastic; there were

no extra-ocular palsies; and pupillary reactions were normal in all respects. No laboratory examinations were made, as the patient was transferred in a few days to the Friend's Asylum, Frankford.

BRAIN STEM AND CEREBELLUM OF A FIVE MONTHS' FETUS. DR. WALTER FREEMAN.

The brain was that of a fetus 20 cm. long, calculated to be about five months old. The child lived for about three hours after natural birth, although during the greater part of this time no respiratory movements were observed. There had been an initial faint gasping cry. The reflexes were not tested.

The whole head was amputated and hardened in alcohol. Later the brain was removed, the pons was cut off above the level of the fifth nerve, and the parts were embedded in paraffin. Serial sections were cut and stained by various methods. Myelin sheath and neuroglia stains were unsatisfactory, but cell stains, and nerve fiber stains by the silver-gelatin diffusion method gave good results. From each 100 micron stratum three sections were studied. The reconstruction was made by projecting the sections on clean 4 x5 inch photographic plates, and then embedding these plates serially in paraffin oil.

External examination of the brain showed: the sylvian fissure was uncovered; a trace of the rolandic fissure could be discerned; on the mesial surface the calcarine and callosomarginal fissures were quite distinct; the cerebellum was small, its fronds poorly developed; the pons and medulla appeared relatively large.

In the sections it was found that the lateral hemispheres of the cerebellum were unequal, the left being the larger. The external granule layer was well shown, the purkinje cells were distinguishable only occasionally. No darkly stained nerve fibers were seen in the subcortical white matter. The nucleus dentatus was of relatively large size, the left one being the more fully developed. The ganglion cells were small and poorly differentiated. A few black nerve fibers could be seen running close to these nuclei in the roof of the fourth ventricle.

In the brain stem some of the nerve fibers stained intensely black with silver, while others remained brown. The cranial nerves, dorsal columns, median fillet, lateral fillet, corpus trapezoides, the fasciculus solitarius, fasciculus longitudinalis medialis, the restiform body and the roots of the trigeminal nerve, took the silver well, whereas the pontile and cerebellar fibers and those of the inferior olive remained pale. The pyramidal tracts were brown throughout.

The different groups of nerve cells also showed variations. The cells of the cranial nerve nuclei, especially the motor portions, were relatively well developed, possessing chromatin bodies, intracellular neurofibrils and blackened processes, while those of the olivary bodies, cerebellum and pons appeared rounded and did not show the internal structure. The globoid cells near the mesencephalic root of the fifth nerve contained no pigment. This mesencephalic root was well developed, but the cells appeared immature.

The nucleus ambiguus and superior olive were very prominent. The former consisted of well developed multipolar cells with intertwining processes. The nucleus could be traced from the lower medulla to the motor nucleus of the fifth nerve, with which it seemed to fuse.

This is not intended to be a detailed study of the structure of the brain stem, but rather to suggest the possibility of using nerve fiber stains on serial

sections, and to suggest that this stain may be useful in conjunction with myelinization studies to determine the development of various tracts in the brain.

NEUROSYPHILIS IN THE AGED. Dr. A. E. Bennett.

This paper will be published in full in a later issue of the Archives.

HIGH GRADE CHOKED DISKS IN EPIDEMIC ENCEPHALITIS. Dr. William G. Spiller.

Three cases were reported of epidemic encephalitis in which a high degree of choked disks made the diagnosis from brain tumor difficult. Dr. Spiller referred to the literature on this subject. The paper will be published elsewhere.

CLINICAL FEATURES OF EPIDEMIC (LETHARGIC) ENCEPHALITIS. Dr. James Hendrie Lloyd.

In the British report on encephalitis lethargica there are references to more than 1,500 articles, written by 1,200 authors, during the years 1917 to 1921 inclusive. The *Revue neurologique*, 1921, contains an index of articles on this disease that fills several pages. It is not to be denied that there is a tendency today to diagnose too many things as encephalitis. We have doubtless all observed instances of this snap diagnosis. I have recently heard of an instructive instance: A patient was sent to a large psychopathic hospital with a diagnosis of manic-depressive insanity; this was changed provisionally to epidemic encephalitis; but a more careful and scientific study in the hospital led to the diagnosis of tuberculous meningitis, which was confirmed by finding tubercle bacilli in the spinal fluid and by the necropsy.

The differential diagnosis between encephalitis and brain tumor may present difficulties which have been recognized by many. A stuporous state is often seen in brain tumor; and this, when associated with paralysis of a cranial nerve, might cause doubt. Choked disk is not a safe criterion. A. S. Hamilton says that, while choked disk is very common in brain tumor and uncommon in encephalitis, it is at least sufficiently common in the latter disease to make it unreliable as a differentiating sign. In the case of a woman at the Philadelphia General Hospital, six months advanced in pregnancy, the symptoms of a brain affection began last September. There have been headache, slight fever, stupor, and now a complete left hemiplegia with motor aphasia, and choked disks. But the spinal fluid is clear and shows only one lymphocyte. The combination of left hemiplegia with motor aphasia, and slight facial paralysis on the right side, is puzzling, and suggests lesions in each hemisphere, which would be more likely in encephalitis than in tumor. I think, nevertheless, that the absence of lymphocytosis is against encephalitis.

Papilledema has been reported now in a number of cases; sometimes these cases are extremely acute, and in one such case there was paralysis of the left facial nerve (Naccarati: *New York Medical Journal* **116**:326, Sept. 20, 1922). Formerly there was a tendency to deny that choked disks ever occurred in this disease, but this denial was based on insufficient observation.

Among the various cranial nerves, the seventh nerve seems to suffer oftener than I had supposed. There is at present in the Methodist Hospital an Italian girl, aged 7, who has had fever and somnolence for more than a month, and now has a complete peripheral palsy of the right facial nerve. She has a lymphocytosis of 175, and the fluid is under high pressure. There has been

diplopia and the left sixth nerve is paretic. It was not possible to test the fifth nerve, as the child cried and did not cooperate. Tubercle bacilli have not been found. It has been thought to be a case of encephalitis. The question is whether the high lymphocyte count in such a case is a reliable ground for diagnosis; also whether tuberculous meningitis can be excluded. There is no ear disease. Dalhern and Grunspan report a case of facial paralysis in lethargic encephalitis (abstracted in *Revue neurologique* **28**:420, 1921).

Of all cranial nerves the third is probably the one most frequently involved—then the sixth. Among the more rare phenomena have been paralysis of associated movements of the eyes, including convergence, and paralysis of accommodation, such as is seen after diphtheria. Paralysis of associated movements of the eyes may indicate either a midbrain or a pontile lesion—but I have seen paralysis of the associated upward movements in a pseudobulbar case due to syphilitic lesions in the lenticular nuclei. French writers have reported a case of hemiparesis of the tongue (Niclot, Cusset and Roubien: *Lyon médicale* **52**:453, May 25, 1920) in encephalitis.

There has recently come under my notice the case of a middle aged woman who was taken with symptoms suggestive of encephalitis: headache, slight fever, diplopia, somnolence and very active myoclonia. These movements were persistent in the muscles of the neck, shoulders, arms, chest and abdomen, and were the most conspicuous symptoms. There was also extremely rapid respiration as high as one hundred to the minute. There was at first a rather high lymphocytosis in a clear fluid. The patient was in the Methodist Episcopal Hospital under the care of Dr. Reiff. No focus of infection was discovered. She passed into a stuporous state with symptoms of meningitis, and a day or two before death developed edema of the lungs. Three days before death, the spinal fluid became cloudy and the cell count was above 2,000, the mononuclear cells predominating. Cultures were negative. The day before death, the fluid was still cloudy and the cell count was reported as 6,000 with polymorphonuclear cells predominating. A necropsy unfortunately was not obtained. I am unable to offer a satisfactory explanation of the case. The clinical appearance in the early stage, especially the extreme myoclonia, somnolence and diplopia, was that of encephalitis, but the later reports from the laboratory seemed to throw doubt on this diagnosis, although nothing better was suggested. There seems to have been some secondary infection; the fluid became purulent, and we might even suspect an abscess of the brain or a purulent meningitis. But an abscess of the brain, so far as I know, does not cause such active myoclonic movements as this patient presented.

A recent French writer (Alaize: *Revue neurologique* **27**:1188, 1920), calls attention to the risk of hasty diagnosis, and mentions superior polioencephalitis and acute bulbar palsy as affections that might be mistaken for lethargic encephalitis. But the identity of these affections is at best problematic. I think it more important to bear in mind syphilis of the brain stem. I have seen very acute cases of this kind; and with the present tendency to jump to a diagnosis of epidemic encephalitis, a mistake might easily be made.

The sequelae of encephalitis may be late in making their appearance. A young married woman, just before her second confinement, complained of seeing double and was unduly somnolent. She passed through her confinement normally and was discharged from the maternity wards of the hospital at the end of a month, apparently well. Two months later she was brought to me with active myoclonic movements limited to the left leg; later they appeared

also in the left arm. She gradually became better, but at the end of almost a year she is still not well. She does not present, as yet, a parkinsonian appearance.

This form, in which the movements are confined to one side, is apparently not uncommon. We have such a case at Blockley in a young woman aged 21, in which the parkinsonian syndrome is present only on the right side; it might be called the hemiplegic type, because there is evidently some weakness, and the lower half of the face is involved. Two French writers (Sainton and Schulmann: *Revue neurologique* **28**:361, 1921) report a case of the "forme hémimyoclonique" in which the movements in the lower half of the face presented a different rhythm from that in the limbs, and they inferred that this "asynchronism" indicated that two different centers were involved. But, in a disease that invades so many parts at the base of the brain and in the brain stem, it is not surprising that irregular and bizarre forms should appear. There has also been observed an alternating type, in which there was myoclonia of the upper limb on one side and hemispasm of the face on the other side, with a paresis of the ocular muscles on the side of the facial hemispasm (Roger and Aymés: *Bulletins et mémoires de la société des hôpitaux de Paris* **44**:689, 1920).

I am not satisfied that harm may not be done by spinal puncture in some cases, especially when it is often repeated. I once referred, before this society, to the case of a child who died of tuberculous meningitis; spinal puncture had been done daily and, postmortem, blood was found effused beneath the pia in the lumbar region. Some recent writers have referred to what they call a "sterile meningitis," which they claim is set up by the Swift-Ellis treatment for syphilis, and which they think promoted the penetration of the arsenic (Mills and Vaux: *Archives of Neurology and Psychiatry* **9**:468, April, 1923). Whether something like this can be set up merely by lumbar punctures, is, I suppose, another matter.

DISCUSSION

DR. F. X. DERCUM: The initial symptoms of encephalitis lethargica are generally vague and not characteristic. There may be a sense of illness, weakness, headache, general pains, chills, loss of appetite, nausea, vomiting, invasion of the respiratory passages; there may be symptoms suggesting a catarrhal cold, bronchitis or even bronchopneumonia. On the other hand, in many cases all so-called prodromata are absent.

Among the earliest symptoms are to be observed: pupillary phenomena such as paresis of accommodation, dimness of vision, diplopia, partial ptosis; the light reflex is commonly preserved, though both Italian and German observers have noted an Argyll Robertson pupil. In a very limited number of cases true amblyopia and even true amaurosis may be noted; very rarely blurring of the edges of the disk with tortuosity of the retinal veins and pallor of the temporal half of the disk, perhaps a slight hyperemia or a slight neuritis. Retrobulbar neuritis or even a frank double optic neuritis has been observed, sometimes, though rarely with hemorrhages. Cranial nerve palsies may be present, e. g. of the seventh, eighth, ninth, tenth, eleventh and twelfth nerves. Perhaps there is in addition, at first, a little apathy or a slight confusion, perhaps insomnia and headache and later somnolence. This somnolence consists, in the vast majority of cases, merely of sleep; sometimes this sleep is very profound but it only exceptionally suggests coma or stupor; usually it is quite possible to arouse the patient and have him comply with various instructions such as to open his eyes, protrude his tongue and at times to sit up in bed or even to get up and walk. When insomnia is present, this is usually associated with excitement, at times with confusion or delirium, and very rarely with convulsions.

In regard to other and later motor phenomena, the motor sequelae, such as parkinsonian rigidity, catatonic fixations, choreiform, clonic and athetoid movements, tremor and irregular voluntary movements, the rarer hemiplegias, monoplegias, contractures and epileptiform convulsions, time will not allow full consideration. Further, many of them are so well known as to form quite common clinical pictures in our hospitals.

Fever is commonly noted, though this fever is rarely high and is irregular in its course—99 to 102 F.; one of my cases for a time had a temperature of 104 F. Little change is shown in respiration and pulse.

The paralysis agitans sequelae, the symptoms referable to the pallidorubral tract, are quite common. Very rarely, however, pyramidal involvement is indicated by the presence of the Babinski sign. The tendon reflexes on the whole are but little modified; they may be moderately increased. At times symptoms of meningitis are noted. It is worthy of comment that cerebellar symptoms are very unusual.

Marked sensory phenomena are not a feature of encephalitis lethargica. On a few occasions various paresthesias have been observed: formication, sandy feeling, a sense of tightness or tension; very rarely hyperesthesia, painful in character. Sometimes a pain suggesting a root pain and, somewhat more frequently, the pain of neuritis is observed. Palsies suggesting diffuse or transverse lesions of the cord are occasionally observed. At other times the symptoms point to the involvement of special columns as in two cases of my own, which suggested ataxic paraplegia. The spinal fluid is not characteristically changed. Regarding spinal fluid examinations in encephalitis lethargica, they should be made with caution.

Complete recovery is rare; and it cannot be said in a given case that the danger of recurrence has passed away until a long time has elapsed. Not infrequently I have noted, in cases with recovery, slight and sometimes marked, persistent pupillary phenomena. A large number of patients die in the early period of the disease.

Dr. William G. Spiller: Stupor in epidemic encephalitis has occurred in my experience, and it has not always been a terminal or even a late symptom. It is true that usually the lethargy is peculiar, in that the patient may be aroused easily and soon falls again into his previous condition, but I have seen patients in stupor so intense that it was impossible to arouse them. The diagnosis may then be difficult. It has always seemed peculiar to me that the cerebellum so frequently escapes, indeed I have not been able to study a case pathologically in which the cerebellum was intensely implicated in the inflammation. Pains in the limbs may be an early and severe symptom of encephalitis.

Lumbar puncture has not seemed desirable to me, because I do not believe it usually has therapeutic results and possibly it may increase the inflammation by lessening the intracranial pressure.

Dr. F. X. Dercum: The matter of spinal tapping should be very carefully considered. I have, as a matter of routine in my hospital cases, tapped for purposes of record and diagnosis, but I have often been afraid that we would withdraw too large an amount and that the diffusion of the infection would be favored by a too great reduction of the intradural pressure. Dr. Gordon's case recalls an experience of my own in which three spinal tappings, performed after intervals of several days, were invariably bloody. The blood was clearly not adventitious but was due to exudation into the spinal fluid.

Dr. John H. W. Rhein: Recently, I saw an unusual symptom in a case of encephalitis lethargica at the Delaware Hospital in Wilmington, in a young

man aged 23, who, after an attack of encephalitis lethargica, developed the parkinsonian type of sequelae together with weakness of the left side of the face. In addition to this was the symptom to which I wish to call special attention, namely a constant smacking of the lips, which was continuous during his waking hours, a symptom that one sees before uncinate fits. This smacking of the lips, which might be termed the uncinate syndrome, is one to which I have not seen reference in the literature.

Dr. Alfred Gordon: I want to relate three interesting cases, two of the so-called hemorrhagic type of encephalitis. Two cases commenced with mental and myoclonic phenomena; one patient died, but before death developed hematemesis. The second case developed buccal and nasal hemorrhages and we also found a hematoma in the quadriceps femoris on the right side. The third case was interesting from the standpoint of sequelae. The patient was a boy aged 12 or 13, whom I saw two years ago with typical encephalitis. He improved and after a period of six months, during which he appeared in perfect health, he developed laryngeal phenomena. He had suffocating spells in which he tried to expel excretions from his throat, but was unable to do so for a time; he was unable to breathe, and jumped off the chair.

Dr. Charles S. Potts: In reference to Dr. Dercum's remarks about loss of sensation, I remember one case that presented a complete thalamic syndrome, i. e. hemianesthesia, hemianopia and slight motor weakness. At first I thought the patient had a tumor, but she developed mental symptoms and was sent to the Pennsylvania Hospital where she recovered. There are some symptoms I have noticed that are not connected with nervous system; one is purulent conjunctivitis, which I have seen in several cases of undoubted encephalitis. I have also seen a skin eruption resembling that of measles. I have observed patients with choked disk; in one case I thought the patient had a tumor, but the subsequent history showed it to be encephalitis. I have noticed that the type of symptoms has seemed to change in different waves of this epidemic. The earlier cases had more pronounced cranial nerve symptoms than are now seen. Then we had myoclonic and parkinsonian types. A peculiar feature of earlier cases was that they did not complain. Now a large proportion of patients complain of headache. Of course, a few of the earlier cases had root pains and headache, but at present the larger proportion complain of headache.

NEW YORK NEUROLOGICAL SOCIETY

Regular Meeting, May 1, 1923

E. G. Zabriskie, M.D., *President, in the Chair*

SUBDURAL EFFUSION AND HYDROCEPHALUS. PRESENTATION OF A RECOVERED CASE. (Illustrated with Lantern Slides.) Dr. Wilder G. Penfield, (by invitation).

The case is that of a boy who developed evidence of increased intracranial pressure after otitis media and had become blind before admission to the Presbyterian Hospital. By means of cranial pneumograms, pressure determinations, and chemical and cytological study of the fluids, it was determined that there had been an effusion of fluid into the subdural space, and that, perhaps

secondary to this, there was a moderate degree of ventricular dilatation. As a result of successive dural punctures, vision returned to a large extent and the other evidences of cerebral compression disappeared. The speaker suggested that subdural effusion would explain the not infrequent association of otitis media with papilledema.

DISCUSSION

Dr. C. A. Elsberg: The case is interesting from a number of standpoints and is certainly of extreme rarity. Subdural effusions are rare, except after trauma, and an effusion as a result of an inflammatory process is certainly very rare indeed. The roentgenograms showed very beautifully the extent of the subdural effusion and also that the ventricular system was open. The case is also interesting from the standpoint of the blindness. For years I have been telling students that, if an individual is blind from papilledema, vision can never be restored. I have tried to save some vision by early operation, anywhere from four to twenty-four hours after the complete loss of vision had occurred, but have never been able to do so. As a result, I was accustomed to say that a patient once blind from papilledema was always blind. Very recently, however, I have had a patient who was entirely blind from papilledema, but in whom considerable vision returned after removal of fluid by ventricular puncture. Dr. Penfield's case is another of this kind. Therefore, I think my opinion as to the hopelessness of loss of vision from papilledema will have to be revised. Dr. Penfield's case shows also how much information can be obtained from a careful study of the roentgenograms after air injection; he is to be congratulated on the careful study he has made.

Dr. I. Abrahamson: It is likely that, in this case, the blindness was due not to the papilledema but to collections of fluid covering and affecting both calcarine areas. The child appeared to have hemianopia at the present time.

Dr. W. G. Penfield: It is difficult to be sure whether or not hemianopia is present in a child of this age. His mother has noticed that he holds objects up to one eye for inspection in preference to the other. The corresponding optic disk is less pale than that on the other side. I have assumed that this rather than hemianopia explains his movements.

ADDRESS OF THE PRESIDENT ELECT OF THE SOCIETY. Edwin G. Zabriskie, M.D.

In this present day world of constant endeavor to increase efficiency in all directions, but chiefly along the lines of production, we at all times encounter the efforts of the medical profession to contribute its share in the work, not only by improving the health of the individual but also by preventing disease. That we, as neurologists and psychiatrists, come in for a heavy share in the responsibility of preventive medicine can not be doubted if one stops to consider that to-day there are 250,000 patients in hospitals for mental diseases in this country; that 50,000 are admitted to these hospitals annually; that the cost of maintenance for those suffering from mental diseases is about $75,000,000 each year, while the economic loss to the United States each year on account of mental diseases is over $200,000,000. These figures do not include the vast number of feebleminded, whose relative frequency in the state of New York alone is 1 to 200 of the population; nor those rendered economically unproductive by reason of pathologic personality traits, chronic instability of the nervous system, psychoneuroses or epilepsy. Neither is it necessary to more than mention in passing those affected organically through occupations in the different industries of the land.

Before the war, our most intensive thought was applied chiefly to the problems of prophylaxis, treatment and custodial care of the insane, the feebleminded, the delinquent and the criminal. Years of patient effort had succeeded in enlisting the sympathetic cooperation of public minded men of diverse occupations and intellectual training, for the purpose of attacking these problems, because of the social economic wastage they represented. A very comprehensive program for intensive study of the situation throughout the country was inaugurated by the National Committee for Mental Hygiene and gratifying results had already been obtained. The entry of the United States into the war, however, interrupted this program temporarily, but its loss was more than offset by the magnificent opportunity to arouse the attention of the community at large to the great numbers of young men eliminated during the hurried assembling of soldiers because of pathologic constitutions or temperamental instability as well as organic nervous disorders, which rendered them unfit for military service. We are now beginning to profit by this lesson, for we realize as never before the almost staggering proportion of unstable individuals in this country, many of whom never reach custodial care, but who nevertheless represent a tremendous decrease of steady production, who, at least in episodic reactions, are potentially insane or are potential criminals. These facts have been accorded due significance in the economic and etiologic problems of neuropsychiatry and are furnishing a still greater stimulus for the prevention of disease.

One might say that it was hardly necessary to formulate so obvious a truism that the proper place to begin the investigation of the preventive side of the problem is during childhood, since science teaches us to search the very beginning of all morbid processes for their causes. This has long been the practice in the study of insanity and of criminology, especially since we have undertaken the investigation of personality make-up, as a glance at the treatises on mental diseases and the studies in juvenile delinquency and truancy will confirm. But now an imperative demand for a more searching investigation of the milder conduct disorders and maladjustments of childhood, the nervous child with tics, speech defects and other evidences of instability becomes manifest, and to-day we see an extensive movement under way in many widely separated parts of this country, which embraces the study and treatment of all functional nervous disorders of childhood, and covers a field the limits and possibilities of which are almost boundless. Problem clinics, mental hygiene clinics for children, school clinics, clinics connected with the courts are springing into existence from coast to coast, and we can foresee the foundation laid for a composite picture of the abnormal individual from childhood on, which will perforce include the anthropomorphic, physiologic, personality and emotional make-up, the environment, and all other factors which might influence the development of the individual.

Some psychologists even are beginning to recognize that other factors than intelligence quotients are necessary for the complete understanding of the individual. Thus, in 1914, Wallin suggested that "Just as soon as a child shows evidence of abnormality, incorrigibility, etc., he should be examined by a psycho-educational clinic which should also afford a medical, hereditary and sociological examination."

The schools furnish a wonderful opportunity for study of these abnormalities either by the group method or individually. The observations of A. J. Pillsbury that 90 per cent. of criminals begin their careers as truants shows what a wealth of material we have practically at our door. On a conservative estimate, from 2 to 4 per cent. of retarded children in the schools are idiots, imbeciles, morons, border cases, epileptics, neurotics and psychoneurotics. From 15 to

30 per cent. grade all the way from border line or seriously backward to merely dull or slow progress pupils. Fully one third are pedagogically retarded when measured by age-grade standard, and about 2 per cent suffer from some form of speech defect. As there are approximately 6,000,000 pupils in the United States, the full significance of these figures becomes apparent to those of us who participated in the army eliminations.

Some one has said that one of the most striking, if not the most important, lesson of the great war was that temperamentally the human race had not changed in any essential feature since the very earliest times. There is much evidence to support the statement and truly we seem to find the same savagery, wanton destruction, wholesale discarding of personal responsibility and mass hysteria, among soldiers and civilians alike, that characterized all former conflicts. Furthermore, each nation displayed the emotional reactions that had characterized it since its inception or, in other words, ran true to its national form. If the above statement contains the elements of truth, as I surely believe it does, one must turn for its explanation to that elusive combination of so-called primitive instincts, unconscious strivings, inhibitions, impulses, moods and emotional traits that contrive to form what we are pleased to call personality, character or individual make-up. Although long ago Herbert Spencer uttered his belief that the whole of life is an adjustment, a continuous adjustment of inner to outer relations, it is only within relatively recent times that sociologist, psychologist and psychiatrist have turned from the study of groups and their classifications to intensive work on the individual and his reaction to environment. Thanks to the illuminating and clarifying studies of Hoch, Freud, Janet, Bleuler and others, we now have at least a working method of approach to the problems of personality make-up, although it is not the purpose of these remarks to do more than point out some of the current ideas. To be sure they are chiefly founded on behavioristic reactions, at least from the viewpoint of MacDougall, who, as the late lamented Southard puts it, has begun to study man's inner relations as they touch society, and from the viewpoint of the sociologists who are endeavoring to carry society back to certain springs of action in the individual mind. To the MacDougall school, character is the result of habit formation or instincts which are amenable to self control and self determination. The psychanalysts, while dealing chiefly with psychopathology, look at personality in terms of sex determinism, conflicts, sublimations, repressions, unconscious strivings, organ representation and the like.

A glance at the most recently compiled endocrine charts will show that a definite place is accorded emotional traits, psychic variability and sexual characteristics, whether they be inversions, heterosexual or hermaphroditic features. Some of the more fanciful of this group of specialists have gone the length of constructing personality types; thus we have: The pituitary type of person who is aggressive, precocious, calculating, self contained; the pituitary inferior: mentally dull, apathetic, sluggish, with poor self control, emotionally unstable, easily discouraged; the hyperthyroid: restless, talkative, liable to anxiety states and vasomotor instability; the hypothyroid: dull, apathetic, prone to all grades of depression, thymic; the morally irresponsible with a tendency to drug addiction, homosexual practice, suicide; the gonodal: introverted, shut-in, sexual and emotional infantilism or the eunuchoid state. One even attempts to link the fundamental instincts with the internal secretions. Thus we read that the two most important instincts which, in the complexity of their sublimations have created most of the institutions of society, viz., the maternal, (social and creative) and intellectual curiosity, (constructive and acquisitive), are tendencies connected with proper function of the pituitary.

In closest relation to the thyroid are the instincts called, by MacDougall, self display and self effacement, exaltation, boasting, exhibitionism or depression, self reproach, etc. Thyroid activity is also expressed in sensitivity, i. e., the ability to discriminate between different grades of sensation and energy. If poor memory goes with the kind of thyroid inactivity seen in cretins, we may deduce that good memory is associated with thyroid activity; it works by increasing the conductivity of the brain.

Again we find exponents of endocrine theories ready to make use of the ideas of Freud, as may be seen from the following quotation of an enthusiastic endocrinologist. "Now all kinds of possible conflict emerge. The child is still bisexual depending upon the amount of internal secretions. The dominant adult of the family, by far the most important of the external factors stimulating or depressing the tendencies of the child, usually possesses a fairly fixed ideal of monosexuality which he or she unconsciously seeks to impose on it. A feminine mother will try to make her son like his father, or, if she dislikes him, like her own father or a favorite brother. A masculinized mother will tend to make a sex object out of the son, i. e., feminization, but the boy on the side of his internal secretions may be definitely masculine, i. e., after adolescence he would be (if the vegetative-endocrine mechanisms created by his mother's personality had not slipped into the inside track so to speak). As a consequence, continual subconscious conflicts between two sets of sex reactions will disturb or ruin his life. So an infant may start out with a fairly balanced endocrine equipment, and yet he may end as an inferior, insane, criminal, or failure, directly because of conflicts between himself as one sex type, and his obligatory associates of another mixed sex type. This applies also to the mother-daughter, father-son, and father-daughter relationship."

One of the essentially novel ideas more or less common to the conception of personality just mentioned, is the idea of determinism and mutability. Those overturn with one sweep as it were the ideas of an immutable personality make-up that might be molded to better adaptation and more harmonious reactions, but still retain its fundamental characteristics unchanged. That we can teach individuals with bad conduct disorders, with bad situation reactions how to overcome their difficulties, how to prevent future recurrences is well known to everyone. That this can be accomplished more readily in childhood is patent to everyone dealing with this phase of life, but is it possible to materially alter the fundamental traits themselves? Some endocrimologists believe it is possible, and one has said that the determination of the endocrine type during childhood and the prediction of future personality type is one of the developments of the future confidently to be expected.

At all events we are entering a period of research in childhood which should constitute the most extensive biologic laboratory experiment the world has ever seen. It will need many years of painstaking, patient collecting of data before the results can be known and unfortunately most of us present will have passed on without more than a conjecture of what they will be.

At a small gathering not long ago it was my privilege to listen to a highly diverting semi-humorous outline of plans for the establishment of a "parentorium," i. e., an institution, controlled by the state, to which parents having difficulty in the management of their children could be committed. The basic idea of the scheme was a reformatory one which had in view inculcation of a proper sympathetic insight into the real difficulties of childhood. Fantastic as the idea may seem, it nevertheless strikes deeply at the root of many conduct disorders in childhood, as anyone familiar with mental hygiene literature must know. The

instance of a mother's despair over the incorrigibility of her little girl, to whom she herself made promises of candy, clothes, etc., as inducements to secure necessary trips to the doctor or dentist or to do something equally unpleasant, but with no intention of fulfilling such promises, and even allowed the child to see her pilfer money from her sleeping husband's clothes, is by no means uncommon. Locking a six year old child in a dark closet for half an hour is no way to cure him of his fear of the dark; nor is the practice of holding the thumb of a four year old baby against a hot stove a safe method of correcting thumb sucking. The literature on delinquents abounds with comments on unintelligent handling of children through lack of proper insight, example, or energetic measures sufficient adequately to carry out recommendations made by competent persons. Frequently the home environment is at fault from physical inability on the part of parents who may be frank neurotics, psychoneurotics, psychopaths of one sort or another, who are thereby not only totally inadequate to handle the situation, but, on the contrary, are often the most important factors in the creation of these situations. Imagine for instance the possibilities for the production of unhealthy traits in a little girl of eight years whose home life is the constant scene of quarrels between a father who is easy going, possesses no ideas of discipline, and accedes constantly for the sake of peace and quiet to the whims of a mother who is profoundly egocentric, headstrong, a day dreamer, hypersensitive and periodically addicted to morphin. The case reports of the Judge Baker foundation abound in such instances merely in delinquents. Our own experience shows that much can be done in clearing up these disorders without thought of altering the personality.

In 1905, in the public schools of Los Angeles, special classes were started for persistent truants. The boys were provided with adaptable men teachers and with curriculums more closely adapted to the life interest of the boys. They were given a type of school work that appealed to their interest and was adapted to their varying capacity. In 1912, there were nine such classes the notable results among which were the following: (1) No boy was suspended from the special classes, the practice of suspending or expelling boys from public schools practically ceased; (2) the average attendance for these classes for seven years was 99 per cent.

The truancy work of the juvenile court was practically abolished. Before the classes were organized all persistent truants were arrested and haled before the court. In 1905 to 1906 there were 56 such cases; in 1906 to 1907 there were 30 cases; after that, never more than three a year and one year none at all. Now the schools handle the truants more economically and effectively. If these boys had only been studied and recorded in the light of modern methods, think what a magnificent start on the path of experiment we might have had.

THE WORK OF THE DEMONSTRATION CLINICS CONDUCTED BY THE DIVISION ON THE PREVENTION OF DELINQUENCY OF THE NATIONAL COMMITTEE FOR MENTAL HYGIENE.

DR. V. V. ANDERSON, (by invitation).

A noticeable swing of the pendulum is taking place in the application of psychiatry to social problems. For a period of years most of the contributions in this field called attention to the striking frequency of mental disease and feeblemindedness among the inmates of penal and correctional institutions, and offenders coming before the courts. Later, it was appreciated that psychiatry could aid institutions and courts in determining the presence of insane

and feebleminded persons, and this sort of service was supplied. Boston Chicago, Philadelphia and other cities began to develop psychiatric clinics for their municipal courts. It was recognized that mental disease and mental defect are less frequent among delinquents than had at first been thought; and that many other conditions, both constitutional and environmental, are also to be reckoned with as determining factors in delinquency. A recent mental hygiene survey in Cincinnati, in which each juvenile delinquent received a careful physical, psychiatric, psychologic and social diagnosis, is significant in its bearings on this question. In a study of the "run of the mine" of the juvenile courts of that city, we found that feeblemindedness was present in only 8 per cent. of the cases; 63 per cent. of the children had an intelligence quotient above 80. We did not feel that inferior intelligence had a very important part to play in the delinquent conduct of these children. Serious mental conflicts, mental maladjustments, emotional complexes, unhealthy imagery, various physical disorders, bad home influences were constant factors. Most important influences in the lives of these children were found in the moral, intellectual and religious atmosphere of the home, in character training, parental control and supervision. These factors we believe have much to do with the formation of character and the development of personality, and here is the crux of the problem so far as the delinquent child is concerned. Eighty-four per cent. of the juvenile court children showed character defects and serious personality difficulties of such a nature as undoubtedly were important factors in their delinquent behavior.

Physical disorders were found frequently and the opinion was justified that the basis and foundation of personality are in the physical organism, and that mental balance and mental health are not possible in the absence of physical health. It was recognized that treatment and ultimate prevention, ever the aims of scientific medicine, seem now to be the dominant aims in the fields of psychiatry. Of the most profound significance in the entire situation is the fact that the great majority of all criminal careers begin in childhood.

In order that the benefit to be derived from the application of the new methods of approach to the problem of delinquency may be made available to juvenile courts, public schools and other agencies in the United States, the Commonwealth Fund of New York City has undertaken a five year program in the prevention of delinquency. This program is probably the most noteworthy undertaking that has ever been entered on in the way of striking at the roots of crime and juvenile delinquency. It provides for a joint campaign on the part of four national organizations, each with a specific task. These agencies are the New York School of Social Work, the National Committee for Mental Hygiene, the Public Education Association, and the Joint Committee on Methods of Preventing Delinquency. Mr. Barry C. Smith, general director of the Commonwealth Fund, is the author of this program. The fund has chosen to concentrate its efforts in the following directions:

1. To develop the psychiatric study of difficult predelinquent and delinquent children in the schools and the juvenile courts; and to develop sound methods of treatment based on such study.

2. To develop the work of the visiting teacher, whereby the invaluable early contacts which our school system make possible with every child may be utilized for the understanding and development of the child.

3. To provide courses of training along sound lines for those qualified and desiring to work in this field.

4. To extend by various educational efforts the knowledge and use of these methods.

"That phase of the program known as section II provides for the creation of a new division within the National Committee for Mental Hygiene, known as the Division for the Prevention of Delinquency. It is charged with the responsibility of determining, through the medium of three clinics, the value of psychiatric service in the study and treatment of conduct disorders in children. Two of these clinics are traveling clinics and move from city to city, giving demonstrations. They are staffed by psychiatrists, psychologists, psychiatric social workers, clinic managers and stenographers, and will remain from six to twelve months in each city for a demonstration of the methods and technic employed in studying and adjusting delinquent and predelinquent children. Such clinics will concern themselves with stimulating social agencies, courts, schools and institutions to carry out the most modern and effective means of treatment. Following the announcement of this service, requests for demonstrations were made by a great many cities throughout the country. These requests came mainly from public officials and social agencies."

St. Louis was selected for the first demonstration and by April, 1922, a clinic had been opened in that city. Cooperation was shown by the various hospitals, city departments, medical men, probation officers and social workers. Soon after beginning the demonstration in April, an advisory committee was appointed composed of judges, medical men, educators, public officials, heads of social agencies and civic bodies in St. Louis. By September, some sixty prominent people, representing business concerns, religious organizations, the medical profession, Washington University, St. Louis University and social agencies had written letters to the Director of Public Welfare urging that he take steps to introduce an ordinance into the Board of Aldermen creating a permanent child guidance clinic in St. Louis, which was accomplished. (The workings of the clinic and a report of several cases were given in detail by Dr. Anderson.)

A monthly follow up form was used by the clinic, with frequent consultation, continual checking up of therapeutic methods employed and further studies of the child. The psychiatric examination outlines and organizes in a systematic way all the symptoms for the purpose of getting at all the underlying causes of psychiatric disorders. It discovers pathologic personalities. It excavates for mental conflicts. It analyzes the mental contact. In short, it seeks to get a picture of the child as a whole, as a living and adjusting personality, and groups the entire study of the case in such a fashion as to map out a scheme of treatment. Psychiatric study of these children brought out a great variety of mental conditions, mental diseases, psychoneuroses, psychopathic personality, epilepsy, endocrine disturbances, mental conflict, feeblemindedness and many and various forms of mental maladjustment which undoubtedly were fundamental factors in the delinquent behavior. The physical examinations disclosed conditions including defective vision, defective hearing, nasal obstruction, diseased tonsils, conditions calling for circumcision, faulty dental conditions, malnutrition, heart conditions, bronchitis, suspected tuberculosis venereal disease, pregnancy, etc.

Summaries.—Two-hundred and fifty records show: 74 per cent. boys, 25.6 per cent. girls; 89.2 per cent. white, 10.8 per cent. black; 59.6 per cent. native born; ages, 3 to 20 years; in ordinary grades in public schools, 58 per cent.; subnormal, 7.6 per cent.; not attending school, 34.4 per cent.

Fifty per cent. had been in court more than once and were considered repeaters; 30 per cent. had been committed to public institutions, many having served repeated sentences. Approximately 87 per cent. gave some evidence of

physical disease, physical defect or physical disorders of such a nature as to require medical treatment. Certain striking disabilities were found in marked contrast to general mental level: 44 per cent. had a specialized mechanical ability; more than 8 per cent. were feebleminded. Other conditions found were psychopathic personality, psychoneurosis, mild personality disorders, etc. The homes of 76 per cent. were distinctly unfavorable to a healthy moral mental development. Sixty-one per cent. had been associated with gangs.

In the study of the 348 children in the "run of the mine" of the juvenile court, the most striking contribution was the great frequency of personality disorders found among children who were repeaters.

Conclusions.—More than half of the children that passed through the juvenile court showed physical and mental disabilities that are fundamental factors in their delinquent conduct. The disposition of their cases, without an adequate knowledge of the social implications of those conditions, merely invites failure so far as the adjustment of the child is concerned. Certainly, intelligent treatment without such knowledge is impossible. While a certain percentage of the children presents serious problems, the great majority of them are not to be thought of in terms of the usual medical classifications, but as examples of childhood difficulties that have their origin in the home, the school, or other situations in which parents, brothers and sisters, teachers and playmates have an important part.

In the particular conduct of the child we find the rôle his personality plays adjusting itself to life's situations. This view of behavior, as a personality reaction in the effort of the child to adjust himself to his environment, by no means excludes consideration of the values to be attached to serious constitutional conditions both inherited and acquired. The importance, however, of feebleminded-ness and grossly abnormal mental conditions is not such as we were led to believe from earlier investigations.

Real progress will be made through a better understanding of the children's personality and the influences from within and without that mold it and make it what it is. Our studies have shown that practically all delinquent children that pass through the juvenile courts are also public school problems. Effective preventive work means the application of more scientific measures in understanding and solving the problems of the children before they get into court, while they are in the school and at home.

DISCUSSION

Dr. Thomas H. Haines (by invitation): These systematic demonstration clinics, which the Division on the Prevention of Delinquency of the National Committee for Mental Hygiene is stationing in various cities, I regard as outposts for the demonstration of what the social engineer, the psychiatrist, can do in the study of the problems of many other boys and girls in our large cities. I do not know how many of you realize that of the 250,000 patients in hospitals for mental diseases in this country, and of the 50,000 who enter these hospitals every year a great number come to these hospitals under very different conditions from those that prevail in the state of New York.

As I think of these patients, I see a post-febrile patient, a woman who has suffered from typhoid fever, pacing up and down the cell of a county jail. Her husband stands outside the cell trying to converse with her. She does not recognize him. She is hallucinating and delirious. While she had typhoid fever her husband could command for her the services of the best medical

talent and the best nursing service in the country. Now because she is "beside herself," because the toxin engendered by the typhoid bacillus had poisoned some of her cortical cells and she is unable to exercise ordinary self control, she is locked up in the county jail and deprived of all medical and nursing service. She is in the care of the sheriff. Should she have been admitted to the state hospital, she must by law have been carried thither by the sheriff unless perchance her husband would have taken her. A large proportion of the female patients in these institutions have been transported by sheriffs. Sometimes such a journey is of hundreds of miles and necessitates a night on a Pullman car or in a hotel. In many of our states it is necessary that the patient be "accused" of being insane and be tried by a jury before a court prior to commitment to a hospital for mental diseases. Six men, court-house parasites, go to the jail to see such a woman and to decide whether she is "sane" or "insane." A more pathetic travesty on justice cannot well be imagined. Yet these anachronistic procedures are in vogue in thousands of communities.

Who is responsible for this lack of appreciation that the mentally ill are sick persons and should be treated by doctors and nurses? Possibly the medical men in these communities, did they but realize the possibilities of their own practice in such cases, could bring about desirable changes.

These clinics are demonstration outposts. They will shed forth light. They will help people to understand what medicine can do for mental disease, as well as for perversions of personality and character. Every one of us has known more than one boy like Gabriel, described by Dr. Anderson. Many of us know that in communities less favored than St. Louis such boys do not get square deals. There are large sections of our country in which there are no such clinics for the analysis of personality and diagnosis of pathologic mental conditions. These carefully organized clinical teams of psychiatrists, psychologists and social workers proceed with the human problems much as does the engineer who takes in hand a problem of irrigation and water power development. He studies the rainfall, the stream flow and the forestation of the region in which he is interested. So these clinics study the organization and the pathology of personality.

I believe that in every city, to which one of these clinics goes and makes a successful demonstration of the service to be rendered by such students of human nature, the service will be made a permanent community asset. I also believe that its influence will reach out over the commonwealth in which the city is located. Such useful analyses of mental pathologies will make their appeal in the country districts. The demonstration that the mentally ill can be helped by doctors more than by lawyers and sheriffs will reach ultimately to all our legislatures. These clinics will be the means of bringing about a decidedly modified and enlightened appreciation of community responsibility in the field of mental maladjustment.

Miss Elizabeth Farrell (by invitation): The type of work described by Dr. Anderson, with modifications that we know to be desirable, has been carried on by the Board of Education of this city for the past twelve years. The work is organized in the Department of Ungraded Classes. The personnel includes psychologists, social service workers, psychiatrists, physicians, and educators. We believe that the problem presented by maladjusted children is one that requires modification of school environment from the point of view of subject matter of instruction, as well as methods of its presentation. Education is a science as well as an art. It offers a field for study and investigation that

engrosses the entire time of scientific men and women who have chosen education as their field of endeavor. The modification of school environment, of school work and of method cannot be properly advised by persons expert in allied fields whether of medicine, social service or psychology. Special education is the proper function of the trained educator, who has available the data which psychology or psychiatry or social service make available on any particular case. This is the major difference between the work carried on by the Board of Education and that described by Dr. Anderson.

The extent of the work in this city will come as a surprise to many of you. During the present school year, which opened on the second Monday of September last, there have been held 470 clinic sessions in various parts of the greater city. The clinics are held in a locality where the number of problem children warrant it. The clinics are of two types. Stationary clinics maintained at headquarters in Manhattan and Brooklyn, and traveling clinics in different localities throughout the greater city. These clinics are maintained for children who present problems of retardation or behavior. These groups include truants, the emotionally unstable, the neurotics, the psychopaths and physically defective as well as many other children who vary from the normal.

The children who are examined at the clinics are selected in two ways. We get many such children as Dr. Anderson has discussed, who present various problems of maladjustment. Their difficulties of course are of long standing. They are a matter of growth. Their management is very difficult because of the prolonged growth which antedates the particular occurrence which has focussed attention on the individual. The habits formed during the period previous to his appearance at a clinic have become an integral part of the personality. To eradicate them is difficult if not impossible. Because of this fact we have developed a technic which brings to the surface children of the type that need attention before the maladjustment has become ingrained. By means of objective measures of entire school units, we are bringing to light the problems of a great many children, at a time when it is relatively easy to redirect energy and effort along lines of positive values. This method of understanding the pupil group in any school unit has been tried in twenty-three of our elementary schools. We find that this study of a school unit brings to light not only those children who are problems to others, the case of Gabriel presented by Dr. Anderson illustrates this type of case, but we get also those children who present problems to themselves. This group conforms to the established order, makes every effort to cooperate, but because of ideals that are unattainable for them, they are waging an unsuccessful battle, they are building up attitudes and habits of failure which they must carry through life. By means of these objective measures and intensive study we are selecting gifted children who, under ordinary conditions in the schools, develop habits of superficiality and dishonesty because school life does not compel them to tap all their resources in order to overcome the obstacles of their environment. We have worked long enough in the schools of this city along the lines suggested by Dr. Anderson, as well as along those lines which we believe to be more comprehensive, to know that it pays. Truancy has not existed in those classes where subject matter and method of teaching have been adapted to the needs, capacities and constructive activities of children.

The principal of a large elementary school in one of the densely populated foreign colonies of the city is responsible for the statement that the problem of bad conduct, which, in the past, has made school discipline an important factor of school work, has entirely disappeared since the pupil group has been

reclassified. She says further that she had not anticipated any such result. It came to her as a complete surprise after the reorganization had been in working order about two months.

My particular contribution to this discussion may be stated as follows: educational clinics must not only attempt to cure children of type, but they must prevent the development of maladjustments. This can be done best by educational clinics which are an integral part of the public school system. Such clinics are regarded by parents, teachers and other workers with children as scientific stations where children are studied in order that delinquencies may by prevented; that success may crown the efforts of children; that their positive self feeling may be conserved; that habits of failure and feeling of inadequacy may not be developed; and finally that the work of the school may be as beneficent as it is efficient.

Dr. L. Pierce Clark: I fully approve of Dr. Anderson's work in all its bearings. Beyond the immediate practical value of Dr. Anderson's results is the highly important effort to solve what may be done to modify human nature either in its inherited or acquired pattern. This larger problem is usually embraced under what we designate as personality, while character is the term that perhaps may be limited to the uses to which this innate personality is put in the process of life adaptations. Personality perhaps may not be changed, or only so slightly as to be negligible, while character lies at the very core of our social adjustment and may be signally changed, with the beneficent results which Dr. Anderson has already reported.

Dr. Sanger Brown II: In correlation with Dr. Anderson's work I wish to speak of some other interesting studies which Miss Theis of the State Charities Aid Association of New York has been making. She has records of about 950 children who have been placed out in homes by that society. These children all came from very poor homes. They were either orhphans or destitute children and their parents were for the most part irresponsible people. From the standpoint of heredity it is very interesting to know how these children have turned out. Miss Theis' findings indicate that despite their bad or uncertain heredity these children are not very different from children from normal homes. Many of them have now reached the age of 18 years and over. There is not a high percentage of insanity among them; there is not a high percentage of mental defect, and there are not many delinquents in the group. Most of them belong to the artisan class and they appear to be industrious and law abiding. This is a hopeful aspect of this whole problem of child welfare in the case of dependent and neglected children.

Dr. S. R. Leahy: We surveyed 1,600 children at the Protectory last summer. We found that, according to intellectual percentage, 85 per cent. were of average intellect, about 5 per cent. were subnormal and the rest were superior. We also found that third offenders were all practically four years or more back in grades. These offenders had apparently not profited by one or two admissions to the institution. Of course with such a large group it was almost impossible to study personality or to get family histories. I think the background has a great deal to do with it. We will have to study and correct this, if possible. I feel that Miss Farrell is correct and that school is the first adaptation; the child is there forced to meet others outside the family; he feels that there are other children who have rights. If he is successfully placed there, it will be a real prevention of further defects. It is only after unsuccessfully meeting

school situations that difficulties develop. I feel that definite and proper placement from the beginning of school life will occupy the proper interest of the child who will fit in very definitely with either of the large types—the superior, average or the retarded.

DR. V. V. ANDERSON: I am sorry I could not have time to read some of my statistical matter. As regards the school as the location for the clinic, I may say that we have one clinic now demonstrating in the school system. This clinic embraces children in the Juvenile Courts or public schools. Thirty or forty came to the clinic because of truancy. In no case did we find this as the most important. As regards Miss Farrell's remark about checking up. We have one clinic for the past year studying schools in a public community. They are studying the school methods of the child and the physical state of every child. A report on this will soon be out which shows the study and checking up of many children. The schools are the location for a clinic. I think the question is that every large city needs clinics in schools; also, the Juvenile Courts need them. In smaller cities it has been my habit to advise community clinics and that Juvenile Court clinics should not be neglected. These have been of real service to a large number of parents who have problem children aged 3, 4 and 5. I think community clinics are ideal for smaller cities. Contrary to what Miss Farrell says, it has been my experience in small cities that parents come in large numbers. It has been my experience that we have had most difficulty to get parents to help on account of their fear that teachers would know private conditions of their life. Our records, however, are always purely confidential.